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OF THE KANSAS MEDICAL SOCIETY

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2 Bayles, T. B.: Medical Forum, Mod. Med. (no. 24) *18*:86, 1950.
3 Gilbert, J. T., Jr., and Moore, F. H.: J. Kentucky State M. A. *48*:308, 1950.

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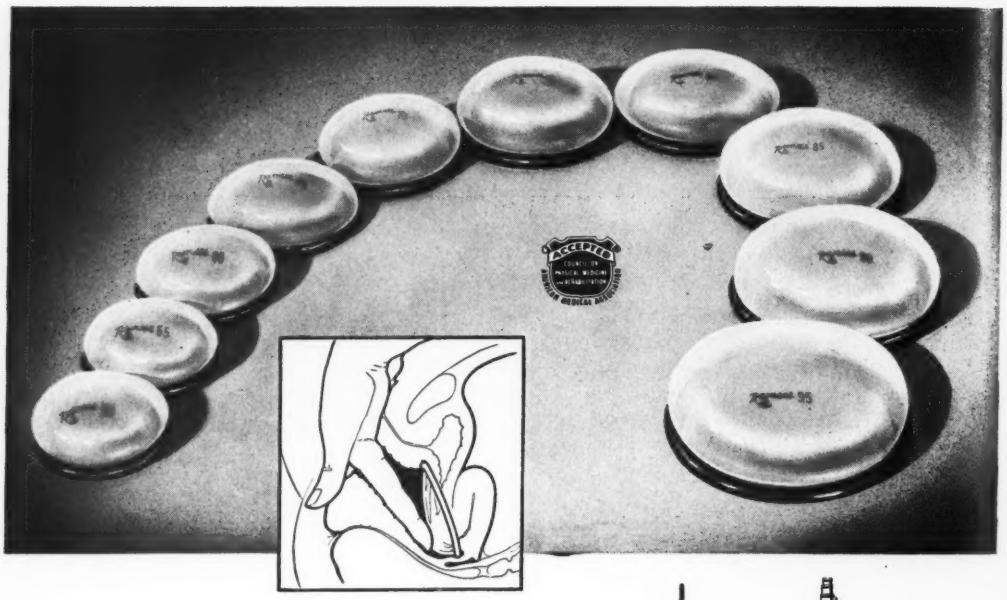

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1. Clark, Le M.: The Vaginal Diaphragm. St. Louis, C. V. Mosby Company, 1938; p. 43.
2. Dickinson, R. L.: Techniques of Conception Control. Baltimore, Williams & Wilkins Company, 1950; p. 17.

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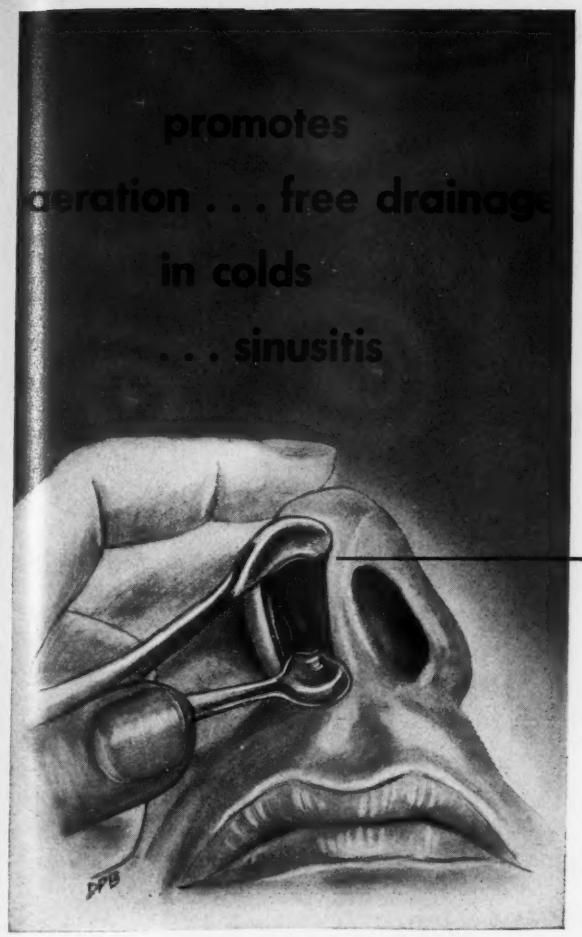

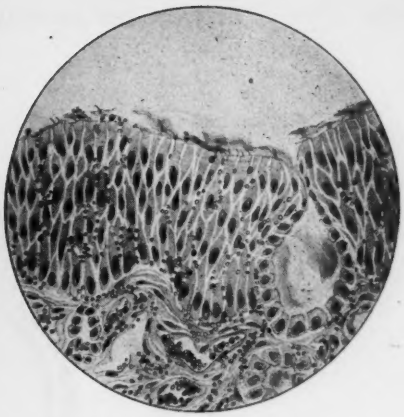

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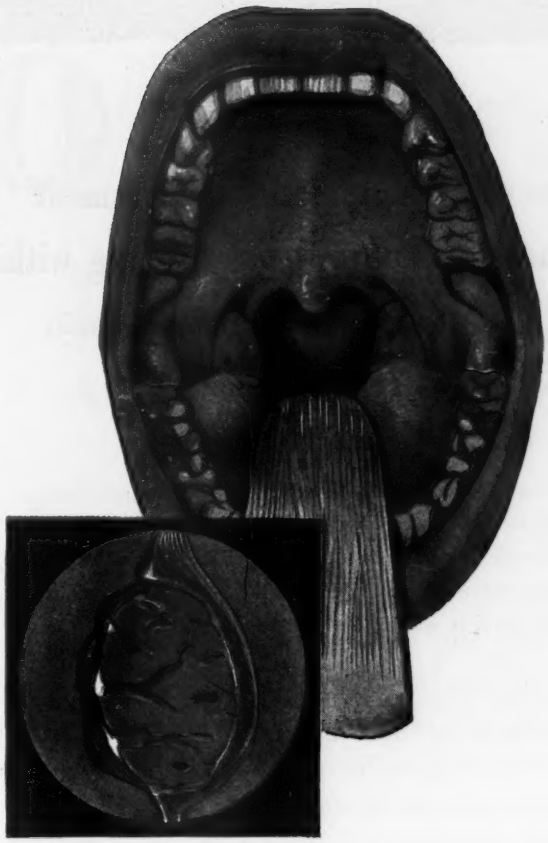

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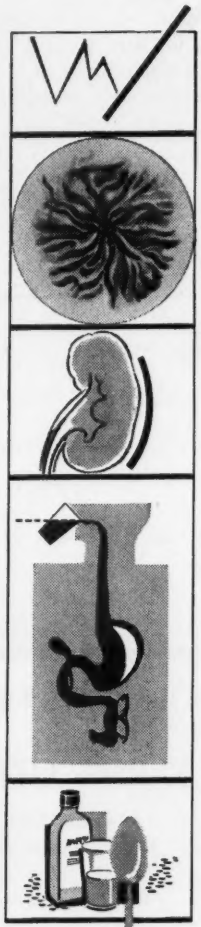

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REVIEW OF ANATOMY OF THE PULMONARY SYSTEM (Illustration Models, etc.)
PHYSIOLOGY OF RESPIRATION
PULMONARY FUNCTION STUDIES AND THEIR APPLICATION
ROENTGENOGRAPHIC METHODS IN PULMONARY DIAGNOSIS
CATHETERIZATION OF PULMONARY VESSELS AS A METHOD OF DIAGNOSIS IN PULMONARY DISEASE (Demonstration)
PULMONARY EMPHYSEMA — THE DISTURBED PHYSIOLOGY — MANAGEMENT
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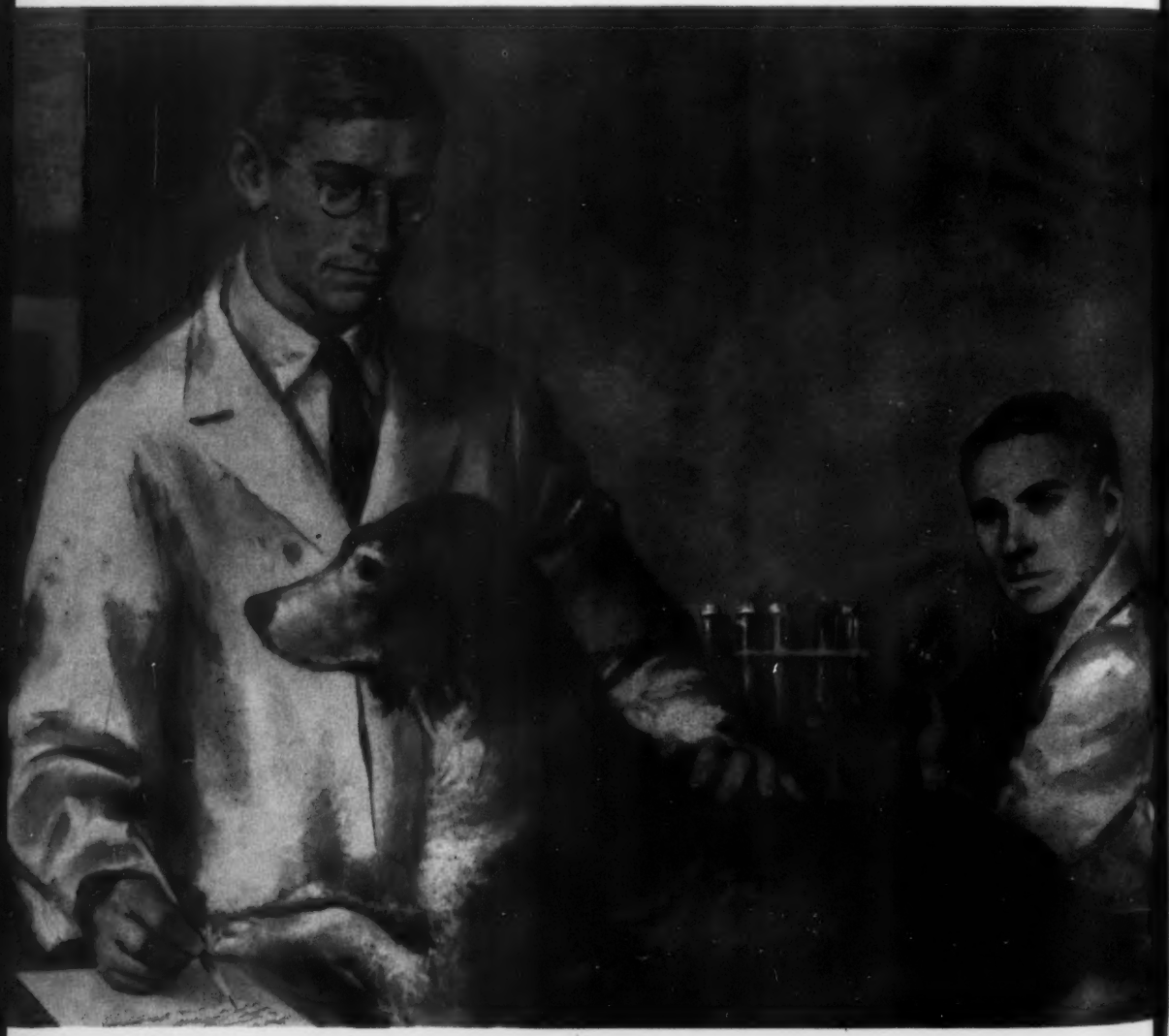

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The Cerebrovascular Accident: A Review of Some Pathological and Clinical Problems*

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Kansas City, Kansas

The term "cerebrovascular accident" or "stroke" is used to denote a sudden injury to the brain or its covering by a disturbance in function of a cerebral blood vessel. This can occur by embolism, thrombosis, or rupture with subsequent intracranial hemorrhage. Embolism by a detached blood clot, gas, air, or fat arises from disease or trauma in other parts of the body in individuals who may have healthy cerebral arteries, and except for the suddenness of onset is clinically similar to arterial thrombosis. The problem of venous thrombosis will not be discussed. The relationship of arteriosclerosis to hypertension and associated vascular disease in the heart and kidneys is so closely bound together that one cannot be considered independent of the other. Arterial disease, due to syphilis, trauma, or infection and arterial hypertension associated with nephritis, urinary obstruction, obesity, endocrine disease, aortic valvular disease, or coarctation of the aorta, belong to the less common causes of cerebrovascular accident and need not be further mentioned.

*Read before Golden Belt and Central Kansas Medical Societies, January 4, 1951.

**From The Department of Neurology, University of Kansas School of Medicine.

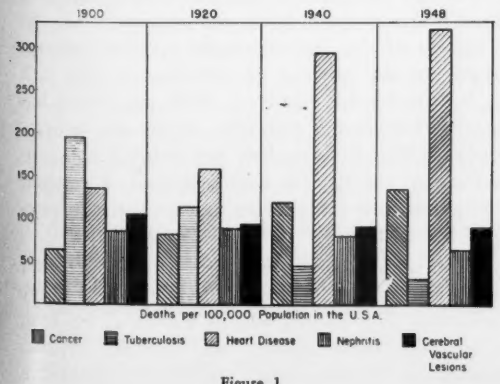

Figure 1

It is interesting to compare some statistics taken from data of the United States Public Health Service regarding some principal causes of death in the United States during the past half century (Figure 1). Tuberculosis has greatly declined as a cause of death, but death from cancer has steadily risen, but not as phenomenally as death from heart disease. It is well known that arteriosclerosis, whether associated with arterial hypertension or not, has a tendency to injure three vital organs; namely, the kidneys, the heart, and the brain. There has been no change in the number of deaths from nephritis and cerebral vascular lesions comparable to the rise in death from cardiac disease. The strain of living is unquestionably greater now than 50 years ago, yet it is not clear why other organs vulnerable to vascular injury in arteriosclerosis and hypertension have not equally suffered. There are many problems about arteriosclerosis that must be solved before the future can answer this question.

Arteriosclerosis is a term that has established itself in medical circles to denote what may be a number of different pathologic processes that have in common thickening and degeneration of the arterial walls. Degeneration of the medial coat in old age with necrosis and calcification, commonly called Monckeberg's sclerosis, is seen in the peripheral arteries. This process seldom occurs in the brain but has been described in the larger vessels of the basal ganglions by Spielmeyer.[1]

The most common type of degeneration of the larger arteries is atherosclerosis characterized by intimal thickening which undergoes degeneration with deposition of fatty substances, and to a much less degree thickening and sclerosis of the media. Splitting of the elastic fibers with hyaline and mucoid degeneration of the connective tissue and eventual calcification complete the picture. The

pathologic change in the smaller arteries and arterioles is of a different character. It consists of thickening of the vessel wall with narrowing in diameter of the lumen. The intima undergoes hyalinization and there is hypertrophy and collagenous degeneration of the media. These two major types of arterial disease vary widely in degree and distribution in different cases, and there may be no parallelism between the degree of arteriosclerosis and functional disturbance.

It has been emphasized by Scott[2] that arteriosclerosis of the larger arteries, commonly seen in elderly people, may give rise to cerebrovascular accidents by occlusion or rupture without any evidence of hypertension. Stenosis or occlusion of the coronary arteries may also occur in such individuals, but renal insufficiency is rare. On the other hand, hypertension and cardiac hypertrophy is associated with sclerosis of the arterioles. Arteriosclerosis of the larger arteries is often combined with this process.

The relationship of arteriosclerosis to hypertension is still an unexplained problem, yet this relationship is important in the study of the cerebrovascular accident. Autopsy studies show that atherosclerosis and arteriolar sclerosis occur together in both hypertensive and non-hypertensive individuals. One is sometimes surprised to see extensive atherosclerosis of the arteries at the base of the brain in relatively young hypertensives. Thrombotic softening in the brain may be found when a cerebral hemorrhage is expected. Conversely, in advanced age hemorrhage may be found in the brain when thrombosis is anticipated.

Moritz and Oldt[3] studied the arterioles in hypertensive and non-hypertensive individuals. They state that there are three views advanced to explain chronic hypertension: 1. That renal disease is primary; 2. that renal disease is a part of a diffuse primary vascular disease; and 3. that hypertension is primary and the renal and vascular changes are secondary to it. It is well known that essential hypertension commonly precedes evidence of renal failure, but it is not certain whether chronic hypertension precedes vascular disease that leads to renal atrophy. The experimental work of Goldblatt[4] directed attention to the relationship of renal ischemia to the pathogenesis of hypertension.

Moritz and Oldt[3] compared the degree and incidence of arteriolar sclerosis in various organs in 100 hypertensive and 100 non-hypertensive individuals. It is significant that the arterioles in the kidney were sclerotic in 97 per cent of the hypertensive cases but in only 12 per cent of the non-hypertensive cases. It has been assumed that chronic hypertension may be the result of primary renal arteriolar sclerosis, either by reflex spasm of peripheral vessels initiated by the ischemic kidney or by retention or elaboration of pressor substances produced by reduced blood flow through the kidney.

Another theory postulates that chronic hypertension may be due to primary arteriolar sclerosis with narrowing of the vessel lumens which produces increased resistance to blood flow. A third theory presupposes that the arterioles are in a state of spasm, thus producing intravascular pressure which leads to degeneration in the arterioles of the kidney and other organs, and that this change in turn increases the severity of the hypertension. The fact that hypertension occurs in some instances without evidence of arteriolar sclerosis in the vessels of the kidney has led to the speculation that the ischemic brain, as well as the ischemic kidney, may elaborate a pressor substance, or by some other mechanism, produce hypertension. For example, hypertension has been produced in dogs by ligation of the cerebral arteries (Nowak and Walker,[5] Fishback, Dutra and MacCamy[6]). The role that the hypothalamus may play in this mechanism is unknown.

Emotional tension and anxiety are believed by many to be a factor in the production of hypertension. The overflow of emotional stimuli into sympathetic stimulating autonomic centers could represent a neural pathway capable of increasing blood pressure, but it is doubtful if this factor alone can explain chronic hypertension in so far as every physician has seen chronic neurotic patients who have persistent anxiety and autonomic excitement for years, yet are quite free of hypertension. The type of arteriosclerosis known as atherosclerosis seems to be associated with a metabolic disturbance, particularly a disturbance in fat metabolism. This has been stressed by Leary.[7] The relationship of this process in man to experimental arteriosclerosis has recently been studied by Altschul.[8]

Despite the gaps in our knowledge regarding basic causes of hypertension and arteriosclerosis, the diagnosis of the cerebrovascular accident presents many practical problems to the clinician. The first to be considered is whether a stroke the patient has just had has followed embolism, thrombosis, or hemorrhage. This is particularly important if one considers that the type of treatment used in cerebral thrombosis may be injurious to a patient with cerebral hemorrhage.

Cerebral Embolism. Embolism can be suspected when a stroke occurs suddenly without premonitory symptoms in a patient who is known to have an acute or subacute bacterial endocarditis, auricular fibrillation, or intracardiac thrombosis, destructive

lesions in the lung, and disease of the aortic wall, capable of releasing thrombotic masses or clots. Once an embolus has lodged in a cerebral artery the symptomatology is similar to that of cerebral thrombosis.

Cerebral Thrombosis. Thrombosis is dependent on disease of the vessel wall, atheromatosis, arteritis, syphilis, or infective state, changes in the coagulability of the blood, cardiac weakness, or low blood pressure, especially in old age. After thrombosis occurs ischemia of the tissues, supplied by the thrombosed vessel, produces nerve cell death, tissue edema, and eventual encephalomalacia. The role that vascular spasm plays as a primary or secondary process is an unsettled and argumentative problem. The symptomatology depends on the degree and site of the lesion, the degree of edema and amount of collateral circulation. Cerebral thrombosis in rare instances occurs from compression of an arterial trunk by a brain tumor, during pregnancy, in exanthems and fevers, and during anesthesia. Steegmann and Feil[9] reported two cases of hemiplegia following cardiac standstill.

Thrombosis occurs most frequently in the middle cerebral artery or its sylvian branches. It occurs only one-third as often in the posterior cerebral artery, and the lowest incidence occurs in the anterior cerebral artery. In the middle cerebral artery, the sylvian branches are involved in about half of the cases, the deep branches that penetrate to the striatal nuclei in about 30 per cent and the remainder of the cases are mixed. Occlusion of the basilar and cerebellar arteries is rare.

Cerebral thrombosis, unlike cerebral embolism, is preceded by premonitory symptoms. Among these are headache, which is frequently localized, vertigo, transient periods of mental confusion, paresthesias or weakness. Convulsions occasionally precede the paralytic signs. Unconsciousness occurs when the area of the brain involved by thrombosis is large. Progression of signs and symptoms may also be evident in individual cases. Goodman[10] has recently emphasized that recurrent thromboses in hypertensive patients may lead to cerebral symptoms, particularly mental disturbances, and this may overshadow any evidence of renal or cardiac damage. Mental confusion also occurs in hypertensive crises which are followed by cerebral edema.

Cerebral Hemorrhage. Hemorrhage into the meninges, brain substance, or ventricles can occur singly or in combination by the rupture of a cerebral artery of large or small caliber. Hemorrhage and softening of the brain following extensive thrombosis of the venous dural sinuses will not be considered here. That condition has been accurately described by Courville.[11]

The most common cause of hemorrhage into the leptomeninges is rupture of an intracranial aneurysm which commonly occurs in the arteries making up the circle of Willis at the base of the brain. This produces a spontaneous subarachnoid hemorrhage, characterized clinically by meningitic symptoms, especially stupor, rigid neck, fever, and grossly bloody cerebrospinal fluid. The aneurysm is congenital, almost invariably in young patients, but may be of arteriosclerotic origin later in life. In individual cases, the lesion is of syphilitic or infective origin. Focal neurologic signs, such as ocular paralysis or other cranial nerve palsies, or more rarely, motor weakness of one or more extremities, precede the symptom of rupture. Severe headache, or pain in the eye on one side of the head, may be complained of just before unconsciousness or stupor. Many of these lesions can now be visualized, if suspected before rupture, by the use of intracranial arteriography.

Hemorrhage into the brain substance has been recorded in almost any part of the brain. It extends and ruptures into the ventricular system, or leptomeninges, in a high percentage of cases. The cause of arterial rupture in cerebral hemorrhage has been a battleground of theory and argument since Charcot and Bouchard in 1868 advanced the idea that miliary aneurysms occur in the cerebral arteries in certain locations and their rupture gives rise to cerebral hemorrhage.

Rosenblath (1918) attempted to explain hemorrhage around cerebral arteries by assuming that there was primary injury to the surrounding brain tissue by the action of tissue ferments. Westphal and Bär thought that spasm of the artery was followed by hemorrhage in the ischemic changed brain tissue. Ricker maintained that cerebral hemorrhage in smaller branches of the vascular tree depends upon a functional circulatory disturbance in which stasis and finally diapedesis of red blood cells around small arteries follow paralysis of vasomotor nerves. Schwartz accepted this view and explained large cerebral hemorrhages by the flowing together of these small hemorrhages. These theories have been reviewed by Bauman.[12]

Beitzke[13] has applied a technique of embedding the clot of the cerebral hemorrhage in one piece and sectioning serially. He has been able to confirm Charcot and Bouchards' original view: that the hemorrhage arises from the rupture of a miliary aneurysm in the artery. This is usually brought about by an increase in blood pressure and by the fact that

the surrounding brain tissue is very soft as compared to the tissue of the kidney or other organs. The aneurysm is formed by local injury of the intima and elastica interna. Arteriosclerosis and syphilis are factors in individual cases. Cerebral hemorrhage may occur as a late complication of head injury, probably as the results of traumatic injury to the blood vessel wall.

The differential diagnosis between cerebral hemorrhage and cerebral thrombosis has been analyzed by Webster[14] and by Aring and Merritt.[15] In general, their conclusions agree. These studies indicate that cerebral hemorrhage is more sudden in onset, immediate unconsciousness being twice as common as in thrombosis. It occurs at a relatively younger age in patients who have a higher mean systolic and diastolic blood pressure level. It is preceded more often by severe headache, vomiting and convulsions. Nuchal rigidity, variations in respiration, and progression of symptoms are more frequent in cerebral hemorrhage. An initial leukocytosis and increased cerebrospinal fluid pressure are found more often in hemorrhage cases. A very important diagnostic sign is the presence of blood in the spinal fluid. This occurs in about 75 per cent of the cases of cerebral hemorrhage. There is no blood in the spinal fluid in over 95 per cent of the thrombosis cases. *In the absence of choking of the optic discs, it is inexcusable to omit the spinal puncture as a diagnostic aid in cerebrovascular accidents.*

The prognosis is much more grave in cerebral hemorrhage. Of Webster's 126 cases, two-thirds died within a week and nine-tenths within a month. In cases of encephalomalacia only one-tenth died within a week and one-third in a month. Aring and Merritt studied 245 cases. They reported death from cerebral hemorrhage in 50 per cent within four days. In thrombosis, death occurred in 28 per cent within four days.

There are two types of cerebral hemorrhage, both almost invariably fatal, that deserve special mention. The first of these is primary pontile hemorrhage. I[16] found in an analysis of 17 cases a disturbance in respiration in every case and respiratory failure in about 65 per cent. Slow gasping respiration, pinpoint pupils, and grossly bloodly spinal fluid are useful diagnostic signs. Death occurred within three to ten hours in 11 of the 17 cases.

The second condition, intraventricular cerebral hemorrhage, cannot be differentiated in most cases from primary pontile hemorrhage. Hemorrhage into the brain substance may precede ventricular rupture, in which case signs of paralysis may be present before the stupor of ventricular rupture begins. After ventricular hemorrhage, there is sudden deep coma, bilateral loss of muscular power, flaccidity or occasionally rigidity and grossly bloody spinal fluid. Some patients have convulsions, usually on the side opposite the ventricle that is filled with blood, or, if bilateral, opposite to the ventricle most distended with blood.

It is beyond the scope of this paper to consider all of the focal signs of brain injury found in cerebral hemorrhage and thrombosis. Although paralysis of one or more extremities may be the most obvious manifestation, it is important to search for defects in the visual fields, change in sensation, or coordination, and if the patient is right handed and has a right sided paralysis, asphasic disturbances. Lesions in silent areas, multiple lesions and lesions of a diffuse character, give rise to psychotic behavior.

Treatment. The treatment of a cerebrovascular accident must be approached as an individual problem. It is an advantage to be familiar with the symptoms and condition of the patient preceding a stroke, an advantage the family doctor may have over the specialist. We cannot be certain, but it seems probable that weight reduction, particularly in hypertension, avoidance of strain and excitement and a more leisurely life may delay, if not prevent, a stroke in some cases. Certainly cardiac reserve, kidney sufficiency, and the average level of the blood pressure are important considerations in treatment. Many arteriosclerotic patients seem to live more happily and comfortably when treated with regular doses of caffeine, phenobarbital, and nicotinic acid, or other vasodilators.

Cerebral Hemorrhage. Once the diagnosis of a cerebral hemorrhage has been made, good nursing care is essential. The posture should be changed frequently, the linen kept clean, and suction must be used to keep the airways open and secretions out of the throat. The nasal tube may be indispensable to administer fluids, drugs, and food in stuporous patients. Therapy is directed towards attempting to reduce intracranial bleeding, keeping the blood pressure and intracranial pressure under control, and supporting cardiac function. In some cases in which the blood pressure is very high, venesection may lessen the strain on the heart. Neurosurgical removal of a subcortical clot has been a life saving measure in some instances. The use of the oxygen tent and hypertonic glucose, or sucrose, to reduce the intracranial pressure helps support the cerebral circulation and protect uninjured nerve cells.

In subarachnoid hemorrhage spinal drainage was formerly widely used. We have abandoned this practice in recent years, and the spinal fluid apparently becomes free of red blood cells and becomes xanthochromic just as rapidly as it did with

spinal drainage. Recurrence of bleeding in spontaneous subarachnoid hemorrhage is a serious complication and is frequently seen in fatal cases.

Pneumonia, urinary infection, and other complications may be avoided, or combatted, by the use of antibiotic drugs which may tide the patient over the critical period to the convalescent stage. Continuous catheter drainage during the stage of urinary incontinence is valuable, just as it is in spinal cord injuries and infections. After the acute stage sedatives may be used, but opiates should not be used in cerebral hemorrhage. Proper consideration of fluid and electrolyte balance is essential. The use of rutin alone, or combined with ascorbic acid, has been advocated to reduce intracranial bleeding, but it is probably of no value.

Cerebral Embolism and Thrombosis. In these conditions the problem of therapy is vastly different. Focal ischemia, which follows occlusion of blood flow, causes rapid injury to the nerve cells, and therapy is concerned with the attempt to restore and maintain the cerebral circulation, particularly in the viable zone surrounding the area of complete ischemia. It has been shown that the nerve cells of the cerebral cortex, striatum, and pallidum cannot survive complete anoxia for a period of over five minutes without undergoing irreversible degeneration. In cerebral thrombosis and embolism, complete ischemia occurs in only a fraction of the area supplied by a given artery. The peripheral areas are protected by collateral circulation of adjacent vessels. Further damage may take place by secondary edema, stasis, and the effect of accumulated metabolites in the ischemic tissues. Nerve cells represent a high metabolic tissue that not only requires an abundance of oxygen but must depend on the glucose in the circulating blood for energy since the cells themselves do not store glycogen.

The shock reaction of a stroke may lower the systemic blood pressure which has been shown by Cobb[17] to be one of the principal factors in maintaining the cerebral circulation. The arteries of the brain are more resistant to change in caliber than vessels in other parts of the body (Loman and Myerson[18]). Stimulation of the cervical sympathetic nerve fibers produces ten times the vasoconstriction in the arteries of the skin than it does in those of the pia mater. Carbon dioxide is the most effective vasodilator of the cerebral vessels. Vasodilatation of the cerebral arteries seems to depend more on chemical stimuli than upon neural mechanisms. The intrinsic control of the blood vessels of the brain seems to depend upon tonic dilatation rather than constriction, and the former is mediated by chemical rather than neural stimuli. Histamine and nicotinic acid are widely used as vasodilators but both of these drugs, although strong extracranial vasodilators, have irregular or relatively mild influence on the intracranial circulation.

Schmidt[19] in a comprehensive study of the cerebral circulation asserts that the vasomotor nerves influence the extracranial vessels much more than the intracranial vessels. He states that evidence is against the concept of cerebral angiospasm from sympathetic impulses, yet vasospasm is still widely accepted as a cause of cerebral circulatory disturbance.

Vasospasm following disease or injury to the arterial wall may occur and may not depend on stimulation of sympathetic impulses from a more remote site. Vasoconstriction can be produced by decreased carbon dioxide and a high oxygen (85-100 per cent) level. Alkaline agents, increased carbon dioxide (5-7 per cent) and decreased oxygen tension (10 per cent) produce vasodilatation. Epinephrine given systemically will increase the cerebral blood flow, dilate the cerebral vessels and increase any tendency to hemorrhage. Histamine increases extracranial blood flow but decreases intracranial flow. Aminophylline, another widely used drug, decreases the cerebral blood flow. Most of the drugs have hardly any vasoconstrictor effect on the cerebral arteries, but vasoconstriction is constantly found in arterial hypertension.

Hyperventilation occurs in strokes from disturbance of the respiratory mechanisms. This tends to decrease the blood flow, but inhalation of high oxygen concentration will do the same thing. Insulin will increase the cerebral blood flow and decrease oxygen consumption. Increased intracranial pressure increases cerebral vascular resistance and decreases cerebral blood flow and oxygen consumption.

Schmidt states that patients with hypertensive vascular disease who have normal cerebral function show normal values for cerebral blood flow and oxygen consumption but they have increased cerebral vascular resistance. In such patients, blocking of sympathetic impulses by various techniques decreased cerebral vascular resistance with the exception of stellate ganglion sympathetic block. This procedure failed to decrease the vascular resistance of the cerebral vessels and failed to increase the cerebral blood flow. In patients with hypertensive vascular disease who had mental deterioration there was a decrease of both cerebral blood flow and oxygen consumption. This can probably be explained by fixed anatomical changes in the cerebral arteries, which would limit the effectiveness of drugs used therapeutically.

A consideration of all of these factors should warn

us of the importance of case selection in attempting to scientifically appraise the value of any type of treatment. Certainly elderly patients with advanced arteriosclerosis, or hypertension of many years duration, or patients with advanced renal or cardiac complications, can expect only some palliation of their symptoms by the most sagacious medical management.

There have been a number of reports[20, 21, 22, 23, 24, 25, 26] indicating favorable results in arterial hypertension following splanchnicectomy and thoracolumbar sympathectomy. Case selection is important for success of these operative procedures. The writers referred to above advise the operation on patients who are not over 50 to 55 years of age, who have normal cerebral function, who have no evidence of cardiac decompensation or severe kidney damage, and who do not have severe retinal arteriosclerosis. A preceding cerebrovascular accident is not considered a contraindication. The patient should, of course, be informed of unfavorable changes that may be produced by the operations, for example, sexual impotence and fatigability.

It has been shown that the absence of retinal arteriosclerosis does not necessarily indicate that the cerebral arteries are free of arteriosclerosis. Alpers, Forster, and Herbut,[27] by careful histologic studies, demonstrated that retinal arteriosclerosis only indicates that the basilar artery may be arteriosclerotic, but gives no indication of the state of arteries and arterioles in the meninges, cerebral cortex and basal ganglions. They found that retinal arteriosclerosis occurs in combination with cerebral arteriosclerosis six times as frequently as it occurs without sclerosis of the cerebral vessels, but the latter occurs as frequently in the absence as it does in the presence of retinal arteriosclerosis.

Stellate ganglion sympathetic block has been used in the treatment of cerebral embolism and thrombosis in recent years. Among a number of reports may be mentioned those of Naffziger and Adams,[28] Searles and Nowill,[29] Scheinberg,[30] and deTakats and Graupner.[31] Scheinberg studied the cerebral blood flow before and after stellate ganglion block and found no appreciable change in blood flow. deTakats and Graupner admit theoretical objections to the value of the procedure, but report that daily stellate ganglion block improved 41 out of 50 cases treated. In view of the fact that they used oxygen, hypertonic sucrose, or concentrated albumin, aminophylline, and anticoagulants, and in view of the general difficulty encountered in clinically evaluating the exact site and extent of softening or the relative collateral circulation in a given case of thrombosis, it would appear to me difficult to evaluate the effect of this single procedure. On the other hand, the theoretical objections to the procedure, on the basis of its failure to increase blood flow in the brain, should not be taken as absolute proof.

We do not know what effect there might be on a lesion by simply opening up the peripheral cranial vessels. The reports of improvement of old cases of thrombosis, weeks or months after the insult by stellate block, are as fantastic as they are pathologically unsound. It is well known that many hemiplegics recover to a surprising degree with ordinary treatment. It is also well known that mental motivation and suggestion may greatly influence the clinical status of individual patients. More evidence on carefully selected and controlled cases is needed before proof of the value of stellate ganglion block can be considered a certainty. A comparative study of results of a large series of cases treated with good medical management, or with stellate block alone, would be of real value.

I am convinced that a large percentage of patients who have strokes do not get the best medical management. The patient with a cerebral hemorrhage is going to suffer from shock and cerebral tissue injury, and his fate will be determined by the site and degree of hemorrhage regardless of the treatment employed. The type of treatment described above must be carried on vigorously until survival of the initial effect is accomplished.

In patients with thrombosis, the same initial procedures as described in the treatment of cerebral hemorrhage are employed, and early ambulation, muscle re-education, and exercise will restore many to useful lives. Before the ambulant stage, contractures of muscles and fibrous ankylosis of the joints of the paralyzed limbs can be prevented by daily passive motion, stretching, and massage. The prognosis, in general, is better in patients with encephalomalacia, but the site and extent of the lesion will determine the outcome. Medical treatment aims towards maintaining good circulation in as much of the ischemic tissue as possible.

The choice of drugs used is usually influenced more by the personal experience of a given physician than by proved scientific principles, and indeed, the proof of the value of a given therapeutic agent is often impossible. This will always be true in a problem presenting so many unknown and variable factors. I, personally, prefer the use of caffeine and ephedrine in patients who are not hypertensive, or in those whose blood pressure has fallen following a stroke. In hypertensive patients, and those with cerebral hemorrhage, I prefer to use aminophylline, because of its general action on the heart and circulation, and hypertonic glucose, or

sucrose, as a 10 per cent solution by intravenous drip. This will tend to prevent the increase of systolic pressure that occurs in increased intracranial pressure. The use of anticoagulants to prevent extension of intravascular clotting in vascular occlusion may promote hemorrhage in dilated capillaries in thrombotic softening and this danger may outweigh any advantage. Reese and Kant[32] recommend aminophylline in doses of 0.1 to 0.2 grams, four times a day, in arteriosclerotic patients with hypertension, especially if they are confused or agitated. They believe the drug has a safe and satisfactory sedative effect superior to barbiturates.

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Cardiac Arrest with Successful Resuscitation

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The incidence of death on the operating table is on the decrease in selective surgery, due mainly to four factors: (1) better pre-operative preparation of the patient in which the nutritive condition, body chemistry, fluid balance and electrolytes are carefully evaluated; (2) use of whole blood transfusions or acceptable intravenous substitutes to replace loss in blood volume and its components during surgery; (3) the great improvements made in anesthesia; and (4) the increase generally in the technical skill and knowledge of the surgeons, especially with regard to cardiac resuscitation.

Respiratory failure on the operating table can usually be handled successfully by the anesthesiologist by endotracheal intubation and establishment of forced ventilation through a closed system with 100 per cent oxygen until the underlying condition corrects itself or is treated accordingly. However, this is successful only if adequate circulation is maintained by the heart, and if this becomes embarrassed due to cardiac arrest or standstill, then the surgeon must re-establish the circulation by the surest and quickest method at his command, namely, cardiac massage. The following case report is illustrative.

Case Report

Mrs. S. B., white, age 20, was admitted to Wesley Hospital for selective cesarean section April 6, 1951; her estimated date of delivery was April 11, 1951. Past history revealed a previous pregnancy that was successfully carried to term, but was stillborn after a difficult and protracted labor with high forceps being used. The patient's prenatal course with this, her second pregnancy, had been uneventful. Ten days prior to admission x-ray pelvimetry had shown a generally contracted pelvis with gross cephalopelvic disproportion, and a test of labor contraindicated. The remainder of the past history was otherwise negative, there having been no previous surgery or anesthesia.

Pre-operative medication consisted of nembutal, grains three, the night before; atropin sulfate, grains 1/100 *1 given 30 minutes before surgery. Spinal anesthetic consisting of 14 mgm. pontocaine in two cc. spinal fluid was given in the fourth lumbar interspace with the patient in the sitting position. Patient was immediately placed in Trendelenburg position with the head elevated on three pillows. After one and one-half minutes, she suddenly became unconscious, respiration immediately ceased, no pulse could be detected at the wrist or neck, pupils were dilated and cyanosis developed. Forced ventilation with 100 per cent oxygen by intermittent pressure on oxygen bag was carried out for two minutes, during which time auscultation of the heart revealed no heart sounds; 0.5 cc. of epinephrine was then injected into the heart muscle*2 with no results. About two and one-half minutes had passed and the condition of the patient was more grave.

The skin of the abdomen was hastily wiped with an antiseptic and the scalpel secured. A single incisional sweep was made from the xiphoid process to two inches above the umbilicus. This one incision carried through the skin, fascia and peritoneum with one stroke and nicked the serosa of the stomach as well. *3 There was no bleeding from the wound edges. The liver was pushed posteriorly, the stomach moved to one side and the tip of the scalpel was then pushed through the diaphragm carefully in the midline (there is a sulcus or mild indentation present to help determine the midline) about one and one-half to two inches below the level of the skin. As the pericardium is fused to the diaphragm along this area, I was in pericardial cavity and a moderate amount of fluid escaped. The diaphragmatic incision was lengthened just enough to permit me to introduce my hand; the heart was found to be small, firm and in systolic arrest. On the first contact with my hand the heart quickly relaxed, gave two contractions and arrested itself again; after three or four seconds I again put my hand on it and, again, the same relaxation occurred, followed by two or three rapid contractions with another arrest. After another pause of three or four counts, I firmly seized the heart and started to squeeze or massage it and, after several massages, the heart suddenly gave a couple of irregular contractions and then established a rapid rate of approximately 160 beats per minute.*4

As the circulation became re-established and

*1 No immediate preoperative sedation of patient with barbital or narcotic drug was given in order to avoid possible depressant effect on the fetal respiratory center. The patient had a calm, stable emotional system and was not in any way apprehensive as she entered surgery.

*2 Although the epinephrine was injected with a long needle into the fourth interspace next to the sternum, no blood could be obtained because there was practically no cardiac cavity as a result of the systolic arrest, hence, the reason for injection into the heart muscle itself. At this time I believe it appropriate to state that I agree with other writers who generally conclude that this procedure is but a method of procrastination in an emergency of this kind and can only serve to delay definite surgery at the cost of precious seconds.

*3 The stomach was greatly distended with oxygen as a result of the positive pressure being applied during the artificial respiration. Before the final closure of the abdomen, the gas was evenly distributed throughout the intestinal tract.

bleeders appeared, these were clamped. After an interval of one minute it was felt that the cardiac crisis was under control, an acceptable pulse being palpable at the wrist, and that the fetus should now be secured as rapidly as possible. Hence, another incision was made in the lower abdomen and a classical section performed with delivery of a cyanosed infant which responded satisfactorily after a few minutes of oxygen.

The uterine wall and lower abdominal incisions were repaired in a routine manner. Then, fresh gowns and gloves were donned, fresh antiseptic was applied to the upper abdominal incision and the patient was draped for the first time. The diaphragm and the pericardial sac were closed as one layer with six interrupted sutures of heavy chromic. The serosa nick on the anterior of the stomach repaired, the incision was closed in layers with no drains. At the close of surgery the blood pressure was 120 systolic and 80 diastolic, pulse 120 per minute but regular, and the post-operative condition fair.

Postoperative Course

The patient's postoperative course was stormy for the first three days and after that, rapid improvement was made. Sutures were removed from both incisions on the seventh day and the patient was sent home ambulatory with the baby on the eighth postoperative day. Nasal oxygen, blood transfusions, Levine suction and massive penicillin therapy played their part. EKG and chest x-ray showed no significant changes in the heart other than a mild loss of tone with slight decompensation which occurred on the second day and responded rapidly to small doses of purodigin. A severe pericardial friction rub appeared on the third and fourth days and then subsided.

The most remarkable factor about the postoperative course—and this indicates that some brain damage of transitory nature had occurred—was the absolute inability of the patient to apprehend pain. Not once during the postoperative course did she complain of any pain or discomfort even with two abdominal incisions, nasal oxygen by catheter, Levine tube, colon tube and intravenous infusions all going at the same time. Repeated questioning by the nurses and myself could not get her to admit to any pain or discomfort. A month later, in my office, she readily admitted pain from the needle when given a vitamin injection.

*4. The total time from the occurrence of the arrest to the establishment of cardiac contractions is estimated to be three minutes and was spend as follows: two minutes of artificial respiration, 30 seconds waiting to see if the cardiac injection of adrenalin was going to work, 15 seconds to make the upper abdominal incision, incise diaphragm and enter pericardial cavity and 15 to 20 seconds to establish cardiac contractions.

Discussion

Quite a few reports are present in the literature [1,2,3] dealing with the topic of cardiac arrest and its management when present as an operative crisis. By far, the majority of these have occurred during the operation when either the chest or abdomen had already been opened. If occurring during an open thoracotomy, the approach to the heart presents little difficulty; if occurring when the abdomen is open, then the operator must attempt to massage the heart against the sternum by pressing the diaphragm against it with the hand. Not more than 30 seconds should be spent in this method [4] and then, if by that time no contractions are present the diaphragm must be incised as described above and direct cardiac massage started. If the surgeon is working through a lower abdominal incision, as in a pelvic procedure, it would be better to make a separate high abdominal incision in order to approach the diaphragm rather than extend the previous incision clear to the xiphoid process as must be done in order to gain adequate exposure of the diaphragm.

Only a few cases have been reported, however, in which the cardiac arrest occurred before the beginning of surgery and was treated with successful outcome. Here one must choose between the abdominal approach or the thoracic approach. For the general surgeon not experienced in thoracic surgery and often working in smaller communities where open-drop ether, spinal or intravenous anesthesia are the rule and no facilities for endotracheal anesthesia are immediately available, the abdominal approach has much to commend it, both by the rapidity and ease with which it can be done and because the general surgeon will be operating in a more familiar field. Furthermore, the presence of a pneumothorax, as would necessarily be induced by the thoracic approach, without endotracheal anesthesia is to be avoided if at all possible, as the resultant embarrassment of pulmonary ventilation due to collapse of the lung adds to crisis already present.

The mechanism by which sudden cardiac arrest with respiratory failure occurs during the administration of the anesthesia, be it spinal, regional, intravenous or inhalatory, is a matter of conjecture but probably represents an anaphylactic reaction by a hypersensitive individual.[5] Most cases reported have been cleared of the stigma of faulty administration of the anesthesia and cannot be laid to any dereliction on the part of the anesthesiologist. As in this case, the dosage, administration, and technique are above criticism.

The question as to how long cardiac massage should be continued is a matter of conjecture; it

appears that in some cases an hour is not too long.[4] During this time such auxiliary measures as injection of a weak solution of epinephrine (0.5 cc. of a 1:1000 in five cc. of saline solution) into the auricle may be of value.

Complete cardiac arrest that persists more than three minutes is usually fatal, and if the patient recovers after this length of time, some permanent brain tissue damage is usually manifest. The preceding statement, while dogmatic, is a good one to govern one's actions by and to estimate the time remaining to do what must be done if the patient is to recover successfully. Wolff,[4] in an excellent paper reviewing the literature, reports a case of cardiac arrest occurring before surgery was started, in which a complete recovery was made after six minutes of cardiac arrest, using the abdominal approach. This case of Wolff serves only to emphasize the exception to the rule of three minutes. In estimating the time of arrest, it is terminated by the time that active cardiac massage is begun—not when the heart begins its own voluntary contractions again. It has been shown that a systolic blood pressure of 60 mm. mercury can be effected by cardiac massage.[4] The success of any method depends upon speed, and the surgeon must be psychologically prepared for such an emergency by having given the matter much previous thought. A decision must be made quickly and converted unhesitatingly into action.

Summary

A case of cardiac arrest occurring before the start of surgery is reported in a case scheduled for delivery by section, with successful cardiac resuscitation within three minutes and the subsequent delivery of a live infant by section immediately afterwards.

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Diverticulitis of the Sigmoid as a Surgical Problem*

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Diverticulosis of the colon is not an uncommon condition. Diverticula, consisting of the mucosal pouches which protrude through the muscularis, have been reported to be present in approximately 10 per cent of the people over 40 years of age. Obesity and constipation are known to be important contributing factors in this condition. The sigmoid contains diverticula in 75 per cent of the cases, and the incidence becomes progressively less nearer the cecum; consequently, it is the sigmoid from which most surgical complications arise.

Rankin and Brown[1] state that roentgenologic incidence of diverticulitis was present in 25.1 per cent of 2,400 cases having diverticulosis. There are probably many mild cases of diverticulitis which are never diagnosed. Therefore, it would be difficult to state what percentage comes under surgical care. It is known, however, that the sigmoid is the most common site of carcinoma, exclusive of the rectum, in the colon. Rankin and Brown[1] found diverticula, associated with cancers, in only four of 679 carcinomata of the bowel. This was an incidence of 0.6 per cent. Cancer associated with diverticulitis was found in four of 227 cases. This was an incidence of 1.8 per cent. Hemorrhage in diverticulitis is encountered in only about 10 per cent of the cases, whereas bleeding is one of the most frequent signs of malignancy in the colon.

Most cases of diverticulitis may be treated medically; however, complications must be handled surgically. It is a treacherous disease with many diversified complications, therefore a surgical procedure must be devised for each individual case.

Every surgical procedure must be devised around two points: first, a procedure to carry the patient over the acute phase, whether it is the drainage of an abscess, the closure of a perforated diverticula, or a defunctioning colostomy; second, the care of any complications that may be present and the prevention of further recurrences of this serious condition.

It is difficult to classify the surgical complications of diverticulitis. However, with some overlapping, four clinical pathological groups may be listed in regard to surgical treatment. First, the group with localized, non-perforated diverticulitis with a history of lower abdominal pain, low-grade temperature, slight distention, and all subacute symptoms which subside within a period of three or four days. These symptoms recur at intervals.

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Second, the acutely ill patient following a perforation with general peritonitis or localized abscess, all of which is in the absence of complete intestinal obstruction. Third, the acutely ill patient, following a perforation, with complete obstruction of the bowel, most commonly of the sigmoid colon. Fourth, those cases with a long history of chronic leakage and subacute inflammation which involves a localized area of the colon, with invasion of the adjacent bladder or the small bowel, often resulting in fistula formation.

Four case histories have been selected that are fairly representative of the above groups.

Case 1. A 55-year-old obese white female was admitted to the hospital on September 13, 1949. Multiple diverticula of the sigmoid had been found four years previously, through barium studies. Her family physician gave the history of having seen the patient on several occasions, and each time she was suffering with what he thought to be diverticulitis. Her symptoms during these attacks were pain in the lower left quadrant associated with some fever and chills. These symptoms would last for three or four days, but would subside with chemotherapy and antibiotics. There was no bleeding from the bowel. However, between attacks she gave fairly consistent complaints of flatulence, constipation, and tenderness with low-grade pain in the lower left abdominal area. Proctoscopic examination revealed no evidence of malignancy. All other examinations were essentially negative. After preparation of the bowel with enemas and chemotherapy, the patient was taken to surgery.

The abdomen was explored through a left rectus incision and was found to be normal, except for several hard and inflamed diverticula of the sigmoid flexure. Since the bowel had been previously prepared, a resection was done with a primary anastomosis of the sigmoid. The procedure was similar to that done in resecting a carcinoma of that area, except for the amount of meso-sigmoid removed. After the anastomosis was completed, a soft rubber rectal tube was inserted through the rectum and was carefully guided past the anastomosis to the splenic flexure. This tube acted as a splint for the anastomosis and for decompression of the colon.

The patient's convalescence was uneventful, and she was discharged from the hospital 14 days after her admission.

This represents a case of known diverticulosis, with diverticulitis for the past two years. The operating of this case may have been questioned; however, it is fairly certain to say that this woman would eventually have had serious complications. With modern chemotherapy and antibiotics, this type of operation does not represent too great a hazard. Hospitalization and expenses are at a minimum where a primary anastomosis may be done. The use of the rectal tube for decompression alleviates the necessity of a temporary colostomy. This certainly is the ideal case of diverticulitis to operate and care for.

Case 2. A 70-year-old male was admitted to the hospital on October 10, 1949. He gave a history of being ill at home for a period of four days prior to admission. His chief complaint was pain and tenderness over the lower abdomen. This complaint had become progressively worse the past two days and was associated with chills and fever. He also gave a history of frequency and dysuria. He had vomited water twice the day before admission to the hospital. His treatment had consisted of a soda enema which did not relieve distress, but he expelled lots of flatus.

On examination this patient was found to be acutely ill. His temperature was 101.2 degrees F., pulse 110, W.B.C. 20,000, with the differential showing a shift to the left. Urine examination was essentially negative. His abdomen was moderately distended, with acute tenderness and rigidity over the lower and left side. Bowel sounds were absent. Rectal examination revealed what was thought to be a mass in the lower abdomen. X-rays gave no indication of obstruction. Because of the physical findings, this patient was explored.

Through a left rectus incision an acute inflammatory abscess was encountered, which arose from what was thought to be a perforated diverticulum of the sigmoid. Penrose drains were inserted in the abscess area. Solutions of penicillin and streptomycin were distributed in the abdomen. A loop colostomy was brought out at the upper margin of the incision. His convalescence was fairly rough; however, with the use of suction, antibiotics, blood and fluids the patient recovered. The colostomy was functioning well when he was released from the hospital 17 days following his admission.

Barium studies done in one month revealed numerous diverticula of the sigmoid. Proctoscopic examination proved negative for evidence of malignancy. About this time he began having some myocardial difficulty, so any further resection of the sigmoid was not deemed advisable. Five months following his operation the colostomy was closed, using local anesthesia. This man was placed on medical management and to this date has been free of any severe recurrence of this disease.

Smithwick[3] states that approximately 25 per cent of the patients who are treated with colostomy alone will have further trouble. However, in this case at the age of 70 and in the presence of a heart condition, further resection was felt to be too hazardous.

Case 3. A 58-year-old male was admitted to the hospital on July 2, 1948. Twenty years previously he had had a gastroenterostomy for duodenal ulcer. Other than some constipation and vague lower abdominal pains, he had been well since. Four days prior to admission he had developed severe lower left abdominal pain and tenderness. His pain and tenderness became more excruciating, associated with chills and fever. He vomited once, and also complained of frequency and dysuria and inability to expel gas.

On examination at the hospital his temperature was 101.4 F., pulse 90, W.B.C. 18,200 and R.B.C. 4,000,000. His abdomen was quite distended with increased rigidity and tenderness over the left side and lower abdomen. Rectal examination confirmed the findings of a mass at about finger tip. X-ray of the abdomen revealed a distended colon with some air in the small bowel. Findings were fairly evident of an inflammatory mass with large bowel obstruction in the sigmoid area. Etiology was undetermined, other than that a perforation had taken place.

The patient was taken to surgery and a defunctioning colostomy in the right transverse colon was done as advocated by Devine, thus completely diverting the fecal stream. A hard inflammatory mass was felt in the lower abdomen but was not broken down at this time. After a rather stormy course under intensive treatment by antibiotics, blood and gastric suction, he recovered. In one month barium studies were made of the non-functioning colon, revealing the typical step ladder appearance of barium filled diverticula. Proctoscopic examination failed to reveal any evidence of malignancy.

In March, 1949, nine months from his first admission, the patient was again hospitalized and operated upon. Through a left rectus incision the involved portion of the sigmoid was resected, making a primary anastomosis. Very few adhesions or mechanical difficulty were encountered, in view of such an inflammatory processes nine months previously. The patient's recovery was uneventful. Two weeks following this resection, a Devine spur clamp was applied to the previous colostomy. As frequently follows this type colostomy, a ventral hernia was present at the site which required surgical repair several months later.

Morton[2] makes the statement that treatment calls for good judgment and patience on the part of the physician and the patient. How true in this particular case, as the expense incurred aside from time off work is tremendous. However, the fact that he is living and well to date justifies a safe prolonged procedure.

Case 4. This patient, a 63-year-old male, was admitted to the hospital on September 28, 1949. He gave a history of being ill at intervals for the past 10 years with vague intermittent abdominal pain, flatulence and constipation. At no time had he vomited or noticed blood in his stools, nor had he had any recent weight loss. One week prior to this admission he became ill at home with intermittent abdominal pains centered around the umbilicus, anorexia, pyrexia of 101 degrees F., and dysuria. His family physician gave him penicillin and kidney medicine and advised enemas. Because of his continued pain, some vomiting and the poor results from the enemas, he was referred for treatment and hospitalization.

His temperature was 100.4 degrees F., W.B.C. 15,200, and there was some rigidity of the lower abdomen with point tenderness to the right and below the umbilicus. The following day, after treatment with penicillin, his temperature was normal, abdomen flat and no masses were felt, but he still had tenderness below and to the right of the umbilicus.

After three days of normal temperature it was assumed that this was a subsiding appendicitis. The patient was taken to surgery, and through a right rectus incision an extensive old and recent inflammatory reaction was found with no free pus. A mass the size of a lemon was present in a localized area of the sigmoid. This sigmoidal mass was firmly attached to the bladder and surrounding small bowel by old inflammatory adhesions. A frozen section of large nodes in the mesentery proved to be inflammatory. The sigmoid was carefully mobilized from the bladder and bowel so the sigmoidal mass could be exteriorized by the Mikulicz procedure, forming a double barreled colostomy through a small left rectus incision. The pathological examination of the resected sigmoid revealed an acute diverticulitis with focal peritonitis. His convalescence was a little rough but no serious complications arose, so on the eighth postoperative day a spur clamp was placed on the colostomy. On October 15, 1949, the patient was discharged from the hospital. Six weeks later, under local anesthesia, the colostomy was completely closed. To date he has been free of difficulty.

The time of this operation was no doubt poorly selected. However, once the abdomen is opened, something must be done. Establishing a proximal colostomy would have been safe with future resection and primary anastomosis. However, that is time-consuming and expensive, whereas the Mikulicz procedure was just as satisfactory for this type case. It would have been only a short time before a fistula formation into either the bladder or the bowel would have been present.

Discussion of Treatment

It is feasible to assume that with the general use of antibiotics and chemotherapy, more cases of interval diverticulitis should be operated upon. Early operation would prevent more of the serious complications, such as fistula formation to the bladder, etc. With adequate bowel preparation during a quiescent period of localized diverticulitis, it would then be possible to do a resection with a primary anastomosis as was done in Case 1. Mortality and morbidity should be relatively low in this properly selected group.

Perforated diverticulum with either localized abscess formation or general peritonitis requires at least drainage, as these people are acutely ill. Because simple drainage is frequently followed by a fecal fistula and subsequent recurrence of the diverticulitis, a defunctioning colostomy is advocated. Smithwick[3] states that operations which neither divert the fecal stream nor remove the involved segment of the bowel were unsatisfactory in over 40 per cent of the cases.

A preliminary defunctioning colostomy is necessary in cases complicated by obstruction or fistula. Establishing a colostomy should precede resection of the involved area by at least six months or a year. A colostomy as advocated by Devine is satisfactory, as this procedure completely diverts the fecal stream.

In those cases where a localized area of the bowel is involved by subacute inflammatory processes, an exteriorization Mikulicz procedure of the involved area is advantageous. These cases are usually found by accident, most commonly confused with appendicitis or gynecologic complaints. The bowel has not been previously prepared by chemotherapy, so a primary anastomosis is not advisable. Formation of a colostomy only necessitates another operation. The use of antibiotics intra-abdominally and parenterally will make the Mikulicz procedure a much more common operation for selected cases.

Summary

1. Four cases of diverticulitis have been presented, each patient being treated with a different surgical procedure thought to be advantageous for that particular case.

2. The advancements in antibiotics and chemotherapy now make resection with primary anastomosis possible. Primary anastomosis is restricted to those cases of interval localized diverticulitis which are in the quiescent phase.

3. Colostomy with or without drainage, in which the involved segment of bowel is not removed, has further recurrences in many incidences. However, this procedure may be advisable in the presence of other systemic complications.

4. A defunctioning colostomy is mandatory in the presence of obstruction or fistula. A waiting period of six months to a year before resection of the involved bowel is advisable.

5. The Mikulicz exteriorizing procedure is advisable where inflammation is localized to the bowel and in the absence of an abscess. The employment of this procedure may be extended with the use of intra-abdominal and parenteral antibiotics. The duration of hospitalization is much shorter than when colostomy is done prior to resection.

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Fourth Annual

MID-WEST CANCER CONFERENCE

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Psychosomatic Problems of the Involutional Period

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Topeka, Kansas

The involutional period, also known as mid-life, or middle age, is the age period from 40 to 60. Prior to this, most people have more or less comfortably invested most of their interests in their work, in their family ties, and in personal achievement. Middle age brings the first awareness to them that all this cannot always go on. The prospect of progressively declining capacities for work, and of less and less opportunity for achieving the goals they have envisioned begins to become apparent.

Awareness of the problem may begin with a hint of the threat of retirement or an illness that actually forces retirement. Often, just at this time, children, in whom much feeling may be invested, grow up and leave the family circle to make homes of their own. For these and other reasons, the realization dawns on most people at this time that they have reached and are passing their peak of efficiency and productiveness. The fortunate ones are those who have developed interests outside their work, satisfying social relationships, and who have the ability to accept calmly the necessity for readjustment, and for changing their way of living. Those who have not these assets and who therefore become frightened at the prospect of losing those things most important to them become ill at this time.

Grinker states that middle age is not fixed, but that somewhere between the ages of 40 and 60, some individuals become old very quickly, while others remain in the prime of mental and physical efficiency. The readjustment or compromise between goals and capacities for achievement that is required in most people at this age is difficult. Some people then react to this problem by denying that anything at all is happening to them. They may often attempt to return to the aggressive and sexual pursuits of younger days. Some well established and hitherto apparently satisfying marriages break up at this time. The picture of the middle aged man pursuing the young girl is so well known that the age of 40 to 60 has been called "the age of indiscretion."

Another reaction in the face of beginning involution is a retreat from these problems and stresses into old age. Of these people it has been said, "Old age is a frame of mind," and Grinker adds, "of the weary and unsuccessful." He states that a person who wishes to decrease his activities because of a sense of failure often retreats into the attitude of old age. When this happens the person seems to age physically quite rapidly.

A third course in the face of the stresses of the involutional period is the development of involutional melancholia, or depression of middle life. Still another is the development of somatic symptoms.

Among the psychosomatic syndromes of importance in middle life are:

1. *Diabetes of middle age:* Diabetes in middle years seems to be directly related to obesity. The altered ability to handle carbohydrates is not due to an endocrine disturbance in the oxidation of glucose, but is due to over-eating. There are people who have tremendous needs for sources of love, many of whom manage to find them. However, in the middle years, there is often a real diminution in the sources of love, affection, or approbation from persons in the environment. As has often been pointed out, love is as much a human need as are vitamins. One of the commonest ways of substituting for fancied or real diminished sources of love and affection is over-eating. The syndrome of the unloved person who eats a great deal is called bulimia, and is well known. The patient who has too high an intake of carbohydrates develops what might be called oral diabetes.

The treatment of this disorder should include better control of the diet. However, it is not possible to control such a patient's diet as long as the patient's feelings of being unloved remain. The physician, therefore, should also attempt to help the patient reorder his life so as to give him more opportunity for friendly relationships. In order to do this, it is necessary, of course, to be familiar with community resources. The physician should see such a patient regularly and can be an important source of affection for the patient himself. He should inquire in a friendly way after the patient's progress, certainly not restricting himself to whether the patient has kept to his diet or not. One should never lecture the patient as to the necessity for remaining on his diet, but rather inquire warmly and interestedly about the patient's success in maintaining the diet, and about his general well-being. The patient should be encouraged to increase his scope of activities that bring him in contact with other people.

2. *Coronary Insufficiency and Anginal Syndrome:* Twenty per cent of the heart disease mortality in the age group of 50 to 59 is due to acute

*Topeka State Hospital.

coronary occlusion. It is at this age, when increases in tension or acute emotional upsets frequently occur, that the neurotic patient with organic heart disease is most likely to suffer a cardiac breakdown. Many patients who have always had difficulty in expressing angry feelings begin at this time to develop anginal pains. It is well known that several years of anginal pains frequently precede an acute coronary occlusion.

Dunbar's studies have led her to believe that patients with coronary artery disease are primarily people who have been aggressive and dominating, who have also had difficulty with authorities, and who have the need to surpass or subdue superiors. They have for the most part been responsible, hard-working people, with few interests outside their work, and people who take authority and do not share responsibility. They generally have little interest in sports and few hobbies. These are the patients who most often are frightened at the prospect of reaching the down-hill half of life. The fear of being dependent on other people, and of no longer being able to accomplish what they had been able to until this time elicits tremendous anger, as well as fear, that they cannot express. They are also, too often, in the position of having no other fields to which to turn their interest and in which they can satisfy themselves with achievement. The dangerous age for these people is from 45 to 55.

One study conducted on patients with already existing coronary artery disease showed that the fewest coronary accidents occur in those who were able to do the following:

1. Make a daily sustained moderate physical effort.
2. Balance their food intake and energy output so that they were neither over-eating nor under-eating.
3. "Practice the hygiene of a quiet mind."
4. Sleep eight hours a night.

I am not certain what these authors mean by "Practice the hygiene of a quiet mind," but it does seem obvious that if we can help the patient with coronary artery disease by giving him support in meeting his daily problems through sympathetic listening or even at times by-helping him make difficult decisions, we can certainly help him to have a "quieter" life. The suggestions about physical effort, food intake, and sleep are excellent ones.

One might summarize this by saying that the physician should help the patient with coronary artery disease to establish better physical and mental hygiene practice. Here, too, it is important to attempt to interest the patient in hobbies and in fields of activity in which he can do a gratifyingly successful job. To order complete retirement for such a patient is unwise. It is, unfortunately, too frequent that the physician strongly advises the middle-aged patient with coronary artery disease to retire. It has been a common observation that death follows soon after, sometimes in a very few months.

3. *Hypertension:* Eleven per cent of people between the ages of 45 and 54 have hypertension, and approximately 25 per cent of all deaths over the age of 50 are caused by essential hypertension. According to Dunbar, hypertensive patients are socially shy people who feel chronic rage, but who have learned to get along with other people by showing a surface of apparent self-control and reserve. They have a marked conflict between the desire to be active and self-sufficient and the desire to be passive and dependent.

As is well known, no therapy has been found adequate for the treatment of essential hypertension. Both surgical and medical procedures have failed to give lasting results. Psychotherapy cannot succeed in returning the blood pressure to normal levels in people who have had the disease for years as patients in the involutional group most often have. The observation has been made that hypertensive patients frequently die soon after they have to go to the hospital. However, hospitalization can be very valuable, if it means a short period of being cared for in a quiet hospital, surrounded by a calm and cheerful staff. These patients should not be told a long time in advance of tests or examinations that are going to be done, or they will get very upset. Doctors' rounds, particularly when several doctors make rounds together and discuss cases at the bedside, tend to upset them. The patient's physician should see the patient alone and in a quietly cheerful and friendly way. He should be free with reassurance and willing to answer questions.

We are all aware of the marked degree of tension that these patients have, and so, often, they are advised to "relax." This advice to relax is very frequently taken as a command by these patients and carried out in a very tense way. I have in mind a patient whom I saw some six years ago, whose blood pressure was very high, something like 240/140. It was felt that the prognosis was poor. In a very naive way I gave this patient the earnest advice that he must relax. The following day a colleague and I were making rounds and we found the patient more tense than he had ever been before. He was almost shaking. When we asked him what the trouble was he said, through clenched teeth, "The doctor says I've got to relax, I've got to relax, I *must* relax."

When hypertensive patients want to talk about their many grievances, and they feel many, the good clinician listens and does not tell the patient that he must not think of such things.

4. *Rheumatism:* Rheumatic pains in the muscles or joints are grouped together under the term rheumatism, which includes joint disease and muscle pains with or without changes in structure. Halliday has done some excellent studies on this group of patients. He feels that independent, upright, and conscientious people, with a high sense of duty, are most prone to rheumatism. As he sees it, they are rigid, stiff, and touchy people and they become ill with the rigidity, stiffness, and touchiness of rheumatism. He noted that some external traumatic event often precedes the onset of symptoms. These patients are sometimes seen with severe arthritic pain, plus anxiety, tremulousness, and frequently some degree of depression. They can't seem to relax. Halliday has also described a middle-age triad of peptic ulcer, rheumatism, and bronchitis.

When one gets to know these patients, one often finds that they feel misunderstood by people around them, and that they have quite conscious fears of approaching old age. I believe that it is necessary to see such patients at regular intervals and to give them sufficient time with the physician to talk about their feelings of fear and depression. The physician, himself, in being friendly and keeping regular contact with this patient, is frequently the best possible treatment.

5. *Peptic Ulcer:* There is a group of people who have excessive needs to be cared for but whose self-respect forces them to deny this and to over-compensate for it by being independent. They often maintain themselves successfully for many years. This denial and over-compensation tends to break down in middle years. The expression of the need may then be, by way of the autonomic nervous system, in the upper gastro-intestinal tract. In a manner of speaking, the needs are being expressed in dyspepsia and later peptic ulcer. However, the personal care that they wish to have is now acceptable to them.

Grinker calls this the "Wall Street Disease," in that it occurs so frequently in driving and aggressive business men on reaching middle age. He pointed out that the wounds to the self-esteem and the reality stresses of the middle years make it difficult for these people to maintain their defenses as they have for so many years. Medical therapy of the peptic ulcer is obviously the first element in the treatment. This alone, however, is not enough. The primary psychological task is to give the patient attention, both at home and from the physician, and in a subtle way that does not conflict with his inability to recognize or accept this need.

6. *Flight into Old Age:* The weary and defeated give up and become old. They suffer many pains, feel useless and discouraged, and often seem to want to die. Many may even develop the physical signs of advancing arteriosclerosis and senile changes, but this is sometimes reversible. These patients must be enticed into fields of interest. It is often valuable to help them make acquaintances of people of the same age, who can share inteersts with them, but who have not this feeling of defeat. It is important that these patients be made to feel that they are able to accomplish something worth while, and this is frequently handled by encouragement to make such attempts, and reassurance that they are doing something. Sometimes through consultation with the family of such patients their external situations can be manipulated so as to give some opportunity for these satisfactions.

7. *Involutional Depressions:* Many of the somatic complaints and symptoms of the middle-aged are really the somatic symptoms of involutional depression or melancholia. This may apply to easy fatigue, lack of energy, constipation, and morbid fears and preoccupations. The fact that the patient is depressed may be overlooked by the physician while he tries to treat the physical symptoms. The symptoms of a depression are well known to all and include the change of mood, the retardation, both psychological and motor, as well as self-accusatory thoughts, and in patients of this age group, very often, pronounced sexual guilt. As soon as the physician becomes aware of the fact that the patient is suffering from an involutional melancholia, he should refer the patient to a psychiatrist. Treatment of the depression of middle life, when instituted early enough, gives a very favorable prognosis. With the disappearance of the depression, the somatic symptoms will disappear too.

8. *The Dementias of Middle Life:* The ages of 40 to 60 bring the organic so-called pre-senile dementias, which are not strictly psychosomatic in the sense we have been using. Alzheimer's disease and Pick's disease, the dementia of Huntington's chorea, and the primary cerebellar atrophies are very likely to occur in this period. Excellent descriptions of these syndromes are to be found in all standard textbooks of neurology. In the middle 50's arteriosclerotic dementias sometimes begin, also. Agitation, memory defects, and deterioration of personal habits should make one wary of these syndromes.

General Treatment Considerations

Weiss and English, in their book on *Psychosomatic Medicine,* mention the importance of the personality of the physician himself in the treatment of psychosomatic illness. In their words, the physician who is to be successful in the diagnosis and treatment of the psychosomatic syndromes must have a real interest in human beings, as well as in

human disease. To a certain extent he must be able to identify himself with the emotional pain from which his patient suffers.

It is not possible to work successfully with these patients if it is necessary for one to get quick results. The physician who does not encourage the patient to remain in his care if the patient does not get well quickly, in a sense is sending the patient to someone who will take care of his problems. They may, however, get to the wrong person—to a quack or an unscrupulous cultist. The physician, then, who knows that he has not the patience to work with these more chronic, slow responding problems, should see to it himself that the patient gets to someone who can competently take care of him.

One should, of course, never brush these patients off as being neurotic old people who really have nothing wrong with them. For one thing, they do certainly have something wrong with them and they need the physician very much. There is another thing—a careful examination and medical study may reveal organic pathology which can be easily cleared up, or which may require hospitalization or the services of a neurologist.

Having done a careful physical study, and having satisfied oneself as to what sort of pathology exists and to what extent, one should then cease dwelling on the physical side of the picture. Excessive dwelling on physical symptoms and unnecessary repetition of laboratory examinations, as has been pointed out many times, tend to "fix" the symptoms and the preoccupation with the possibility of a physical disease to a point where frequently it is impossible to convince the patient that he is not seriously physically ill.

It may often help, after one has the confidence of the patient, to explain earnestly and frankly, without making it sound somehow shameful, that the patient has emotional problems which contribute to, or cause many of his symptoms.

English lists some means of therapy at the command of the general practitioner. These are:

1. Giving examples of how a physiological condition can be caused by an emotional state. Excellent examples of this are the physical concomitants of fear.

2. Analogies can be used to illustrate how emotional tension can, through the nervous system, affect organ function.

3. Patients can be re-educated as to better ways of achieving normal gratifications, that is, ways of having satisfying relationships with other people.

4. Giving the patient an opportunity to speak about his problems in a warm and understanding atmosphere helps to desensitize the patient to his problems.

5. It is important to give the patient encouragement and approval freely.

6. Follow-up interest is extremely important. One should encourage patients to return at intervals in order to discuss their current status. This can be an important means of continuing the emotional elements necessary to help them maintain psychological balance.

Conclusion

The realization in middle age of the fact that they have reached and are passing the peak of efficiency and productiveness, and that with but few exceptions the rest of the route is downhill, brings many people to the physician with depression, anxiety, and psychosomatic illness. The fortunate ones are those who have developed interests outside their work, satisfying social relationships, and the ability to accept calmly the necessity of readjustment. The most favored few are those who, for reasons we do not understand, need not face this problem at all. To those who have not these assets and are frightened at the prospect of diminishing vigor, this period of involution brings pain and illness. Drugs and manipulations alone will not help them; their human needs require attention, too.

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CHANGE OF ADDRESS

Please notify the Kansas Medical Society of any changes in address.

Help keep the mailing list up to date.

PRESIDENT'S PAGE

Dear Doctor:

I wish to report that we have been diligently pursuing an answer to our rather huge and most bizarre problem of finding an over-all solution; which of course must be compatible with the State Welfare Board, the Kansas Hospital Association, the Pharmaceutical Association and those providing medical and surgical service to the indigent. I am proud and happy to advise that each and every participant mentioned above is fully cognizant of the magnitude of this deeper than apparent social problem! I personally solicit from each and every county your continued patience for a short while, for soon I shall provide you with some startling and amazing facts!

The program for the circuit postgraduate course is now completed and has been in your hands nearly a month. Please permit me to urge your consideration and in the final analysis, your enrollment. I would also urge your recognition of the fact that this excellent postgraduate course is sponsored by your Society, your State Board of Health, and your State School of Medicine—it cannot fail!

It is most gratifying and very stimulating to note the acute interest in society problems shown by many chairmen and their respective committees having early meetings and thereby forming their plans early in order to accomplish the maximum during the coming months. Please accept my congratulations!

I am of the opinion I can speak for each of you in saying to Dwight Lawson, Sam Barham and their assistants—Thanks for a job superbly done on the "Physicians' Manual." I wonder how many of us are putting in a portion of time in behalf of the society that is so nobly represented in the issuance of this excellent preparation?

Very truly yours,

EDITORIAL COMMENT

Traffic Safety

Perhaps you know it already—that the 68 Kansas fatalities in August is the largest toll for any one month in the history of this state—that 387 persons were killed by vehicles in Kansas this year up to September 15, which promises to make this a record high—that nationally 37,800 traffic deaths are expected this year, or 3,000 more than ever before—that 980,000 Americans have lost their lives in traffic deaths since 1900.

Perhaps you know that the Kansas Medical Society is cooperating in a Kansas Conference for Traffic Safety Education, where every possible means of reducing this death toll is being explored. A heartening interest is being shown in driver education classes for young people and adults. Safety films are being presented all over the state. Many organizations are sponsoring safety as principal projects and the man-to-man agreements are increasing in popularity.

These, in case you do not know, are voluntary agreements between father and son that each will respect the car for the power it has, each will obey the laws and use courtesy while driving. Similar pledges are available for father and daughter compacts. While such agreements have no legal status they can be effective exactly to the degree that both parties respect and abide by the principles involved.

You know of course of many vagaries within the law that might be corrected, the farce that lies within the driver's license law, the lack of enforcement of regulations that are in effect. You can point to the need of wider, straighter four-lane highways, special truck routes, compulsory liability insurance and many other things that would correct one or another of the myriad present day problems. All these are being studied and worked at from many sources, but these are primarily on the negative side of the question.

The great and abiding problem remains the driver himself, and on that hazard the medical profession and education have a profound dual responsibility.

You know it is human, not mechanical failure that causes most accidents. Dr. Bryon Stockey described it this way. "The most difficult problem in the campaign for safety is neither the road nor the car. It is the human element. Roads can be rebuilt; new principles of car design can be adopted; but the driver's nervous system cannot be made over . . . His reactions within certain limits are fixed. Yet, he too must be controlled."

It is physically impossible for the medical profession to periodically re-examine every driver, nor is the physician at liberty to notify law enforcement officers of patients considered unfit to operate a motor vehicle. Even if both were possible, it would probably bring about as much harm as good.

There are some things that the individual physician can do. The effectiveness of the medical profession's contribution to this field will just about total whatever interest is shown by the individual. Many possibilities are open, but these few ideas may present other possibilities.

The individual physician can endorse and lead in the public acceptance of safe driving principles. He may materially aid the effectiveness of local driver education courses by offering to address the students on why he advocates safety. He may work toward the passage and enforcement of sound vehicle control laws.

But there is also something more he may do. He may advise his patient on the hazards of driving. Many operators are unaware that their physical, mental or emotional disturbances create danger to themselves and others. Some just do not care.

You and you alone are qualified to judge the nature of his illness and how this might affect his driving. If it is his eyes, you know the extent of his handicap. If it involves his reflexes, you are aware of that problem. If you could convince this large body of persons of the risk they take while operating a vehicle, you could make a tremendous contribution in the solution of this problem.

Yes, it takes time and effort on your part, but it would represent one of the great public services that are open to the medical profession. You have the knowledge to solve this problem and the opportunity. You alone are in this peculiar position, and upon your willingness to accept this challenge rests to considerable degree the prospects of reducing the annual traffic toll in Kansas.

Socialized Medicine in Scotland

Mr. R. C. McCormick, manager of the Broadview Hotel in Wichita, recently visited Scotland and England. He wrote the following article on socialized medicine for the *Wichita Beacon*. It is reprinted here with permission from the author.

"Elie, Fife, Scotland.—Socialized medicine was a new experience to me.

"During a golf game I had taken a clumsy swing at a ball and put a severe stitch in my back muscles. I spent a restless and painful night.

"The next day we were to drive to Edinburgh. I had my host call the family doctor and make an ap-

pointment to give me a shot to relieve the pain. It was early morning, before office hours, so there was no delay.

"I called at the doctor's home and the shot was administered. The reply was 'nothing, the socialist government is paying for it.'

"Not only that, he handed me a prescription headed 'National Health Service (Scotland). Executive Council for County of Fife.' He explained that it was a form of analgesic balm shipped over from America, which was exceptionally good for such cases. Just stop at any pharmacy and present the slip. The government would be glad to give it to me.

"He then went on to say that he would be pleased to write me an order for a wig or a set of false teeth if I felt inclined to have either or both. Again, they were 'free.' The government would 'give' them to me. I was a guest of the British government and during my stay, the government felt obligated to keep me in good health.

"I wanted to ask him if he could give me a prescription for a good, juicy 10-ounce steak and a quart of fresh orange juice. Both are very healthful.

"I was frankly puzzled with the whole procedure. I had contributed nothing during my stay in Scotland. I was at a loss to understand how a free wig would help my health.

"Later when I found that the government was soaking the working man five shillings for a five-pound salary and then taking another five shillings from the employer, I wondered why I wasn't offered a beautiful nurse to rub my brow and to accompany me on the balance of my trip. There was enough money coming in for such generosities. Where was it all going? I should settle for a wig!

"Relating this to American dollars. A man with a $14-a-week salary pays 67 cents a week and his boss pays 67 cents a week for 'free' health service. The total is almost 10 per cent of the man's salary. Take 10 per cent of your current monthly salary, put that figure up against $6.00 a month, the approximate cost of Blue Cross and Blue Shield for you and your family in America. The $6.00 a month is freedom. Ten per cent of your salary is socialism. Take the different between the two figures and multiply is by 60,000,000—the number of workers in America. The result is the waste of socialism.

"For example, say you make $250 a month. You and your employer would contribute $25 a month to health under socialism. Twenty-five dollars, less $6.00, is $19. Nineteen dollars times 60,000,000 is $1,140,000,000 waste.

"This is a very conservative example. The national health service of Britain puts out NO figures on cost. It has been fairly estimated that the British government is putting up $3,000,000,000 over and above its outrageous tax on each worker to carry on 'free' health.

"Sooner or later we will be educated to the point where we all realize that God, in His infinite wisdom, created the world so that nothing is 'free'—not even freedom. We must work for everything we have.

"These politicians who say to us, 'I can fix it so that the government will do it for you,' work upon our ignorance and greed. They fly in the face of creation. As long as they remain in power due to our willingness to believe them, they are a living monument to our stupidity and selfishness. Nothing on this fair earth ever has or even will be 'for free.' When you get 'something for nothing,' you either have paid for it, or it is nothing."

What Is Federal Aid?

"What Is Federal Aid?" was the subject of an editorial that appeared recently in *Nebraska Agriculture,* official publication of the Nebraska Farm Bureau Federation. The definition and the editorial follow:

"Federal aid is a temporary expedient that was offered to state and local governments by idealistic bureaucrats at a time when state and local governments lacked the courage to face and meet squarely their own problems of administration. It is a delusion that has gained permanency because state and local governments have not had the courage to make an effort to solve the problems created by the depression years of the thirties and the war economy that followed. Federal aid has always carried with it the idea that it costs no one anything; it is just money that the federal government gives states and local governments out of the goodness of its heart. Don't you believe it.

"You and I and all the other American taxpayers pay dearly for federal aid. Every dime of federal aid is paid for in higher taxes, higher costs of automobiles, higher costs of fodostuffs and everything farmers and consumers buy, because taxes are a big item in the costs of production of this nation. . . . As a matter of fact, federal aid is the most expensive solution to problems faced by state and local governments, because taxpayers have to pay the costs of bureaucratic idealists who tell the states and local governments what share they shall have of the tax funds available and how they shall administer it."

Now is the time to make reservations for the 93rd annual session of the Kansas Medical Society. The meeting will be held in Kansas City, Kansas, April 27-May 1, 1952.

SOCIALIZED MEDICINE

This is the 23rd of a series of articles dealing with federal compulsory health insurance. They are designed to give the physician factual information and reliable data which may be used in the preparation of articles or speeches on this important subject. Additional material will be presented in subsequent issues.

Socialized Medicine in Great Britain

The following release, distributed by the Committee for Constitutional Government, Inc., was written by Cecil Palmer, English publisher, author and journalist, who is one of the most vocal and highly respected opponents of socialism in Great Britain today.

* * *

For more than three years, the British people have endured socialized medicine. Supposedly intended to provide a first-class national health service, the scheme was politically conceived and is rapidly becoming politically administered. It failed to fulfill its promises and obligations because politics and medicine do not mix. The art of healing has nothing in common with the craft and craftiness of vote-catching.

In its evolution from private practice to state control, the profession of medicine is losing its independence, integrity and indispensable privacy in the relationship between physician and patient. A lay bureaucracy impinges itself on medicine. The profession is cluttered with orders, rules and regulations. It is overwhelmed in an avalanche of forms, questionnaires and certificates which reduce doctors to impotence and patients to despair.

For some years, the American public has been invited to accept politically-planned and controlled medicine because, among other things, it is claimed that such a system is imperative to overcome a potential shortage of doctors. But nationalized medicine has not solved the problem of too few doctors in Britain. On the contrary, some of the best men and women in the profession steadfastly refuse to associate themselves with the scheme. Others who gave it a fair trial are deserting it, by premature retirement, seeking other employment, or by emigrating and requalifying for service abroad where the ethics of private medical practice still prevail.

One well-known doctor said upon giving up practice, "I cannot cope with the situation. To make an adequate living, you must have so many patients you can't look after them properly. What worried me most was not being able to prescribe what I thought was good for my patients. Instead, we are supplied with a list of what we are permitted to prescribe, and I found myself thinking not what is good for a patient but whether a particular prescription is on the list. That is bad medicine."

Broadly speaking, there are not enough doctors, nurses, hospitals or clinics to service the Service efficiently. The maximum panel of patients allotted to one doctor is 4,000 and the minimum is 2,500. The average time available to the doctor for both diagnosis and treatment under nationalized medicine is from three to five minutes per patient!

The first estimate of the cost of this so-called "free" medical service was $500 million a year. Despite the fact that patients now make part payments for dentures and spectacles, nationalized medicine now costs British taxpayers roughly $1,960 million a year, an increase of nearly 300 per cent since 1948. Of this expenditure, only $143 million reaches general medical practitioners. When nationalized medicine was inaugurated their per capita fee was a fraction more than five cents a patient, and this scale still applies today when bare living costs have advanced.

According to the Auditor General, Sir Frank Tribe, (and he should know) nationalized medicine has produced a swollen, expensive bureaucracy of non-professional "administrators." He points out that "of the total hospital expenditure, 9.9 per cent goes in doctors' salaries, 23.8 per cent for nurses, and 26.5 per cent represents salaries and wages of 'other officers and employees' including administrative staffs."

Large sections of the British public have become "bottle-and-pill" conscious. We are extending and fostering a brand of petty hypochondria, hitherto the almost exclusive prerogative of the self-pitying few who imagine that they enjoy being ill. The very sick are herded with the very slick and must wait their turn. The old, the dying and the desperately ill are forced by circumstances beyond the control of either doctors or patients to enter a competitive struggle with their relatively healthier rivals.

There are tragically long waiting lists for admission to hospitals. The number is officially admitted to be more than 500,000. There are also at least 10,000 serious tuberculosis victims awaiting admission to sanitoria. At the same time, there are thousands of empty beds because the available professional personnel cannot cope with the demand for its skilled services.

It is not surprising, therefore, that the Association of Municipal Corporations, representing local government in contradistinction to national government, strongly criticized hospital administration. Appalled by weighty evidence of waste in nationalized hospitals, the association declared that "the direct accountability of the local authorities to the

electorate for the expenditure they incur is more likely to conduce economy than the present system."

The blunt truth is that nationalized medicine is facing a financial crisis. Over-burdened British taxpayers now realize that they are paying far too much for far too little. Doctors and patients alike are bewildered and viciously frustrated. They are in temporary bondage to the totalitarians. It is possible that all concerned are waking up to Herbert Spencer's classic generalization that "the ultimate result of shielding folk from folly is to fill the world with fools."

The British people are not fools and cannot be fooled by knaves and so-called "do-gooders" indefinitely. The pregnant point at issue is not free medicine, which is a palpable lie, but *freedom in medicine* which is an eternal, fundamental truth. Nationalized medicine is operative socialism; and socialism begins in the benevolent tyranny of the Welfare State and inevitably reaches its climax in the malevolent, tyrannical Serf State.

Conference on Heart Disease

A conference on heart disease, to which all physicians in the state are invited, will be held on Thursday, November 15, at Winter VA Hospital, Topeka, under the sponsorship of the Committee on Study of Heart Disease of the Kansas Medical Society. There will be no matriculation fee.

The conference will begin at 10:00 a.m. and will be continued through the afternoon. Luncheon will be served at the hospital.

Dr. Edward Massie of St. Louis, associate professor of clinical medicine at Washington University and a member of the staff of Barnes Hospital, will be principal speaker. The program will be as follows:

The Care of the Pregnant Cardiac—Mahlon H. Delp, M.D., University of Kansas Medical Center, Kansas City.

Electrocardiography and Congenital Heart Disease—Ned W. Smull, M.D., University of Kansas Medical Center, Kansas City.

Case of Mitral Stenosis with Autopsy—Robert T. Cotton, M.D., Winter VA Hospital, Topeka.

Problems in Congestive Heart Failure—Edward Massie, M.D., St. Louis.

Thirty-Minute Question and Answer Period.

Some Metabolic Aspects of Coronary Heart Disease—Richard R. Howard, M.D., Winfield.

Psychosomatic Case Report—Paul W. Yost, M.D., Winter VA Hospital, Topeka.

Studies on Pheochromocytoma—Newman V. Treger, M.D., Topeka.

Case of Malignant Hypertension with Autopsy—Samuel Zelman, M.D., Winter VA Hospital, Topeka.

The Use of a Temporary Polythene Shunt to Permit Resection and Replacement of Vital Vessel Segments with Homologous Frozen Grafts—Paul W. Schafer, M.D., University of Kansas Medical Center, Kansas City.

The Committee on the Study of Heart Disease sponsors two courses each year. The first is a three-day course held in Emporia during October, designed to interest those physicians who have had advanced graduate study in heart disease and a considerable portion of whose practice relates itself to that specialty. The second conference, for which the program is given above, is designed for all doctors of medicine interested in the subject of heart disease. Previous conferences have been well received, and the committee hopes that a great many physicians will attend so that this year's program will be of even greater value than those which have preceded it.

Study on Typhoid Inoculation

A study on the effect of typhoid inoculation of women in the child bearing age is now being conducted in the Kansas City area, and information from physicians in other flooded areas of Kansas is urgently requested. A follow-up study on fetal anomalies, to be conducted in about eight months, is also planned.

Data for the study is being collected by Dr. Roy Busenbark, 224 Brotherhood Block, Kansas City, Kansas, who will furnish blanks for reports. Kansas physicians who gave inoculations to women in the age group being studied are asked to supply the information requested.

Accuracy in Laboratory Diagnosis

A promise of greater accuracy in laboratory diagnosis results from a service announced recently by the College of American Pathologists. The group is initiating a program which will enable pathologists who direct laboratories to obtain highly standardized chemical solutions for use in performing tests on body fluids.

Distribution of standardized dextrose and nitrogen solutions to 1450 member pathologists began August 30. By checking solutions prepared by their own technicians against the standard solutions, directors of laboratories can thereby increase the reliability of their medical tests.

This program is the initial step in the development of a comprehensive plan for standardization of laboratory procedures, the College announces.

Case Report from the University of Kansas Medical Center Clinical Pathological Conference

Myocardial Infarction—Embolic Coronary Occlusion

Edited by Glen R. Shepherd, M.D., and Mahlon H. Delp, M.D., from recordings of the conference participated in by the departments of medicine, pathology, psychiatry and neurology, roentgenology, and the junior and senior classes of medical students.

Case Presentation

A. W., a 48-year-old colored female, was admitted April 30, 1951, and expired on May 1, 1951. One hour after admission, while lying in bed, she developed severe pain in the chest with radiation to the neck and both arms. Within a few minutes she became comatose and was brought to the emergency room.

Physical examination showed a well developed, well nourished colored female who was comatose. The skin was cold, moist, and markedly cyanotic, and the extremities were mottled. The pupils were pinpoint in size. Respiration was shallow and rapid. No radial pulse or blood pressure could be obtained. The heart rate was 72 per minute and there was a gallop rhythm present with a grade III systolic murmur at the apex. Moist rales were heard in both bases. The liver was palpable four finger breadths below the right costal margin and was smooth. There was two plus pitting edema of the legs.

Past history revealed a two month illness at age 13 years characterized by fever and swollen tender joints. A similar illness occurred at age 22. A positive serologic test for syphilis was discovered at age 32, following which she received a course of bismuth and arsenic for over a 10-year period. The serology was negative thereafter. At age 32 she was seen with lower left abdominal pain and excessive menses. Pelvic examination revealed a tender, left tubo-ovarial mass thought to be inflammatory. At age 34 she complained of dyspnea, vertigo, and hemoptysis.

During her 44th year she incurred two cerebrovascular accidents with residual paresis of the left arm from the first and partial paresis of the right arm and leg and a moderate speech defect from the second.

She was next seen at age 45 when she complained of vertigo, headache, chest pain with radiation to the left arm, and attacks of nocturnal dyspnea. At this time she had a grossly irregular rhythm and her blood pressure was 180/85. At age 46 she was in KUMC for two weeks with rather severe pain of sudden onset in the left shoulder. Examination revealed in addition to the previous heart findings absence of the left radial pulse and markedly limited left arm motion. A few weeks later she had an episode which started with syncope and headache and was followed by severe abdominal cramps and constipation. The blood pressure at this time was 160/110. She was hospitalized for this illness and while here had several episodes of severe pain in the right chest. She was hospitalized twice in 1950 for nausea and vomiting thought to be due to excessive digitalis.

The menstrual history revealed gross irregularity since age 32 with frequent bouts of menorrhagia. She always had considerable dysmenorrhea. There were nine pregnancies, one full term normal delivery, one therapeutic abortion, and seven miscarriages, the latter in the early months of pregnancy.

Laboratory findings taken from the outpatient clinic records over the past year show the following: Complete blood count, serum sodium, potassium and chloride, and hepatogram all within normal limits. Urinalysis showed a trace to two plus albuminuria. Wassermann and Kahn were negative. Numerous blood cultures negative. NPN 24.1 and 37.5. Iodine—131 uptake—17 per cent.

During the hospital course, aminophylline and coramine were given intravenously. Over a three-hour period, 250 cc. of plasma and two 500 cc. while blood transfusions were given. Oxygen was given continuously. At one time a feeble radial pulse and a blood pressure of 40 mm. mercury could be obtained. Three hours after admission a bilateral stellate ganglion block was performed. The respiratory rate became progressively slower, being eight per minute 11 hours after admission. At this time there were moist rales throughout both lungs. Further respiratory stimulants were given without avail and the patient expired 12 hours after admission.

Dr. Mahlon H. Delp (Chairman): This patient had been under observation for a long period of time. The diagnosis, while not actually mentioned in so many words, should be rather clear. I refer to the primary disease requiring such long constant observation. The signs and symptoms presented the evening of admission indeed were very dramatic. I think there were a dozen people there who were puzzled by this patient's appearance.

Question: Did the findings include a colloidal gold?

Dr. Edwin Slentz (Medicine Resident): Yes.

That was done in October of last year, and recorded as negative.

Dr. Delp: May we have the EKG's.

Dr. E. Grey Dimond (Medicine): The EKG of July 5, 1948, shows auricular fibrillation with a rate that is quite well controlled. There is probably some right ventricular hypertrophy, but it is difficult to confirm it with the number of leads used.

An EKG of June 23, 1950, about a year before her death, is relatively unchanged. The tall R wave noted over the right precordium indicates a large right heart. It is compatible with the auscultatory observation of a mitral rumble.

The electrocardiogram on the day before death shows a pattern that is rather diagnostic and typical. You will notice the fact that no P waves are apparent, that there is an elevation of the RST segment in Leads II, III, and aVf. There is a reciprocal depression in Lead I and aVl. This is a rather typical pattern of a posterior infarction.

Dr. Delp: Dr. Germann, may we have the x-ray films.

Dr. Donald Germann (Roentgenology Resident): The film I have was taken in January, 1950. The heart is not unusually large. It does have some prominence in the pulmonary outflow. The lateral film is a little more interesting. There is a definite bulge in the region of the left auricle. There was definite fullness in the region of the right ventricle. This goes along with what the history and other findings have suggested in the mitral region.

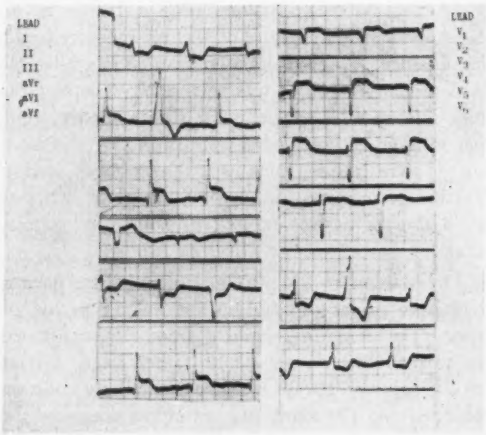

Figure 1. Electrocardiogram taken the day before death, showing the 12 leads in a typically diagnostic pattern. ST segment is markedly depressed in lead I and elevated in leads II and III of the limb leads while the ST segment is depressed in aVl and elevated in aVf. Thus, the current of injury is characteristic of posterior myocardial infarction. The RST duration is consistent with posterio-lateral myocardial injury. Incidentally, the tracing shows auricular fibrillation and frequent ventricular premature systoles.

Differential Diagnosis

Mr. Don R. Meriwether (Medical Student): Even though embolic phenomena as a cause of myocardial infarction are classed as rare, even in rheumatic heart disease, they do occur. Here is a woman who had a history of embolic phenomena for some years past. I think she did have a myocardial infarction resulting from coronary embolization.

Dr. Delp: Why do you think she had myocardial infarction?

Mr. Meriwether: On the basis of the EKG findings and the clinical course. That is, severe pain developed during rest with the typical radiation and cardiovascular collapse.

Dr. Delp: Would you elaborate upon the pathogenesis for this infarction?

Mr. Meriwether: I think it is on the basis of a mitral valve lesion of the heart. The contributory factors were present. These include a long history of rheumatic type heart disease and a history of fibrillation which was still present on admission. It is possible that emboli were coming from the valve but more likely from the auricle.

Dr. Delp: Mrs. Sumner, what do you consider the common cause of death of patients who have rheumatic heart disease?

Mrs. Joyce Sumner (Medical Student): Congestive heart failure.

Dr. Delp: About what percentage of them die of congestive heart failure?

Mrs. Sumner: Eighty to 90 per cent.

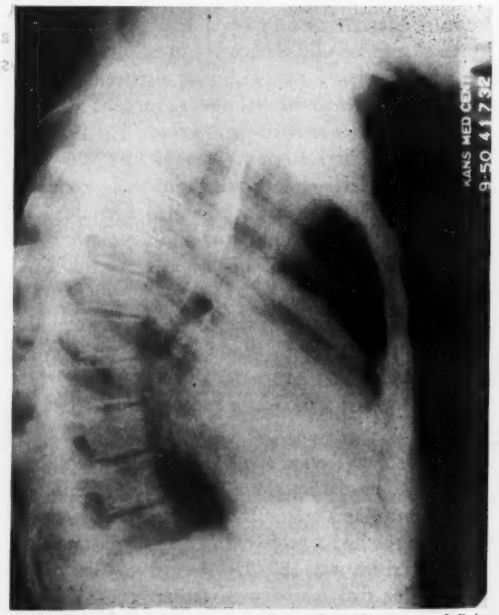

Figure 2. Right lateral x-ray of the chest showing a definite bulge in the region of the left auricle and fullness in the region of the right ventricle.

Dr. Delp: I would like to approach this problem with the staff in just a little different manner. Dr. Wheeler, I would like to know what your differential diagnosis might be, if you had only this patient's symptoms for evaluation. I saw this patient shortly after she had arrived in the emergency room. She was comatose. Relatives said that she had complained of a severe chest pain which came on suddenly while she was sitting up in bed relaxing after her evening meal. She described the pain as radiating into both arms and into the back of her neck.

Dr. John Wheeler (Medicine): The differential diagnosis with those symptoms is considerably limited. Certainly the thing that you would think of first would be anoxic pain from coronary insufficiency or coronary occlusion.

Embolic phenomena with pulmonary infarction is often associated with severe pain but also with symptoms referable to the lungs. Usually these are cough, expectoration of blood tinged sputum, and even collapse.

Dissecting aneurysm of the aorta would not be outside the realm of possibility. Due to the fact that we know reasonably well that she had no aneurysm radiographically, I think that can be ruled out.

I think the diagnosis is obviously coronary occlusion with typical radiation of pain.

Dr. Delp: Dr. Wheeler, do you think this patient's coronary occlusion is on a basis of arteriosclerotic heart disease?

Dr. Wheeler: Yes, sir.

Dr. Delp: Dr. Kaul, this patient had a profound shock. Would that evidence influence your differential diagnosis?

Dr. Philip Kaul (Medicine): I agree with Dr. Wheeler that, if we can believe the history from the family, this patient certainly had typical symptoms of myocardial infarction. Most patients who have a myocardial infarction do not become comatose although a considerable portion of them will have peripheral vascular collapse and shock. However, a certain group of patients with myocardial infarction will become unconscious.

The other thing that certainly should be considered in the differential diagnosis because of this patient's coma is a cerebral vascular accident. The patient had pin point pupils, lack of response, lack of reflexes, and general nervous and vascular collapse. Certainly she could have had either a cerebral embolus or cerebral emboli to one or more areas of the brain.

I think the patient had both a myocardial infarction and cerebral vascular accident due to an embolus. As far as the etiology of the myocardial infarction goes, this patient did have some enlargement of the heart although it is not marked. The patient was an old rheumatic, had had pain in the past which was suggestive of, but not diagnostic of, angina pectoris, had had some degree of hypertension for five or six years. She was 48 years old. Tossing those factors together, still it does not seem to produce enough evidence for myocardial infarction due to thrombosis. On the other hand, you still have to accept that as the probable cause of the myocardial infarction because embolization into a coronary ostia is a rare phenomenon which is rarely seen and rarely reported. Those cases reported are most often associated with subacute bacterial endocarditis. The latter diagnosis, I doubt very much.

Dr. Delp: Are there any features of this case suggesting dissecting aneurysm?

Dr. Kaul: I think it fits it in many respects. The degree of shock and collapse fits the picture of dissecting aneurysm. The pain is not typical of a dissecting aneurysm. Most dissecting aneurysms produce pain that proceeds on up into the head or down the back.

I think perhaps this patient is a little young for dissecting aneurysm.

Dr. Delp: I would disagree a bit with Dr. Kaul. I think the pain is not typical. However, there were some features of the pain that made us think of a dissecting aneurysm momentarily—the wide distribution in the upper segment of the body. The patient's appearance of being in cardiovascular collapse would also fit. Patients with dissecting aneurysm do not usually have such marked fall in blood pressure even though having the appearance of cardiovascular collapse.

Dr. Steegmann, we have not yet adequately considered the comatose state noted here.

Dr. Theodore Steegmann (Neurology): The outstanding things given in the history are first, sudden onset with profound rapid coma, pin point pupils and respiratory difficulty. The pain in the chest has been discussed. Several things can happen in the central nervous system resulting in sudden profound shock with coma. Several things affecting the central nervous system cause a more gradual coma with respiratory suppression.

Opium poisoning could more or less be excluded because of the history of sudden onset, although when you see a patient with pin point pupils and respiratory depression, you should always think of it.

Second, primary pontile hemorrhage can occur in persons of this age and particularly in hypertensive cardiovascular disease. This causes a profound shock, pin point pupils in certain cases, and respiratory failure. I have seen quite a number of these cases,

and in a few of them there was a history of chest pain at the onset. These patients usually become unconscious so rapidly that you cannot always get much history of premonitory symptoms. A large percentage have blood in the spinal fluid. What diagnostic procedures you do in a patient like this have to be modified by how sick the patient is. You're not going to run for a spinal puncture needle in every case you see. In both interventricular hemorrhage and pontile hemorrhage, which is interventricular in a large percentage of cases, grossly bloody spinal fluid is important.

Another thing we think of that might cause respiratory difficulty and small pupils would be the very rare intermedullary hemorrhage in the spinal cord in the cervical region. That might more likely follow trauma, but spontaneously occurring cases have been recorded. The small pupils then are due to blocking of the sympathetic pathways. I assume these pupils were constricted before the patient was given the stellate block. At any rate, hemorrhage in that region would affect respiration because the respiratory mechanisms lie just a few segments higher in the spinal cord.

I would like to mention one thing about dissecting aneurysm of the aorta. That is that those aneurysms also affect the small vessels that originate the radicular arteries of the spinal cord and produce occlusion or a cutitng off of their blood supply. This produces the picture of thrombosis of the anterior spinal artery which in many ways will simulate intermedullary hemorrhage in the spinal cord. It seems to me a rather remote possibility here.

Dr. Delp: Dr. Bolinger did make a completely correct ante mortem diagnosis in this patient, and I would like to have him tell us briefly why he made the diagnosis.

Dr. Robert Bolinger (Medicine): I considered the same differentials that have been mentioned. The striking thing about this patient was, as Dr. Steegmann pointed out, her comatose state. It would be difficult to base any cause for a comatose state outside of some depression to the brain, either chemical, circulatory or traumatic in origin. The profundity of shock could have been responsible for her comatose state for a short period of time. However, a person who is in a state of shock, if placed in the horizontal position with the feet higher than the head, is soon wide awake and appears to be almost alert. This patient was certainly not that.

The fact that this coma continued over a long period of time could be explained only by some disease of the brain itself. With the previous history of two embolic phenomena, I believe if we hadn't had an electrocardiogram on the patient and had merely seen her lying there on the table, we would have simply said that she had a cerebral embolism and that her chest pain was probably due to the same phenomena.

This patient had had chest pain over a long period of time. On the whole the story of pain was atypical. The electrocardiogram showed unmistakable evidence of a myocardial infarction. I think it reasonable to offer a common pathogenesis for the electrocardiographic patterns as well as the central nervous system symptoms.

Pathological Findings

Dr. Ann Pollack (Pathology): The body was that of a fairly well developed, well nourished negro woman. There was a little distention of the abdomen and a little bit of pitting edema of the ankles and nothing else externally.

The heart was enlarged, particularly on the right. The right ventricle was both dilated and hypertrophied. Both the auricles were likewise. The left ventricle, in contrast, was actually small. The mitral valve showed the characteristic fish mouth deformity of severe mitral stenosis. It admitted the little finger with difficulty.

The other valves were not remarkable. Upon opening the right ventricle some hemorrhage was noted beneath the endocardium. This covered an area of necrosis. The apex of the papillary muscle actually extending into the muscular wall itself was surrounded by hemorrhage. This had the gross appearance of being an infarct. But it was in a remarkable location, on the free wall of the right ventricle. Infarcts in the right ventricle are exceedingly uncommon. They represent somewhere between three and six per cent of all infarcts, but they do occur.

The right coronary artery appeared to be dis-

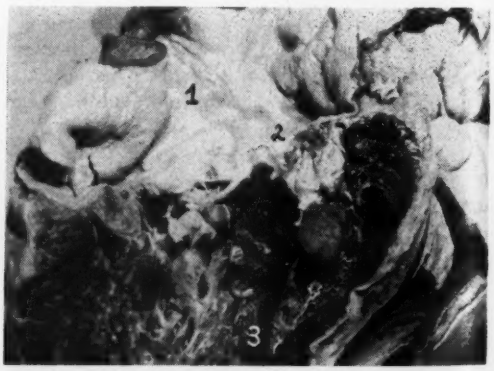

Figure 3. Gross picture of the cut heart. Numeral 1 is on the left auricle, 2 at the mitral stenosis, and 3 on a portion of left ventricle.

tended and in that area of distention was a fresh ante mortem clot. This clot propagated down the distal portion of the coronary artery.

This is a most amazing appearance for an ordinary coronary thrombosis. A thrombosis of any kind conforms to the shape of the vessel in which it is found. However, this thrombosis didn't conform to the shape of it but actually distended it. It looked for all the world as if it had been pushed inward.

The gastrointestinal tract from a portion just beyond the ligament of Trietz through the entire jejunum and ileum, the cecum, the ascending colon, the hepatic flexure, and the right half of the transverse colon were all dilated and presented purple-red discoloration with hemorrhage into the wall particularly in the terminal ileum. That, of course, is the classical distribution for obstruction of the superior mesenteric artery. The superior mesenteric artery also had an ante mortem thrombus obstructing it.

It might be possible, but exceedingly unlikely, to get two simultaneous thromboses, both of them very fresh. The mitral valve did show microscopically a very small adherent thrombus which apparently was the source of the embolus to both vessels.

There were multiple areas of infarction in both kidneys. There were multiple infarcts in the spleen, all of which were old, and several areas of old softening in the brain. There was also chronic passive congestion in the liver with some cirrhosis.

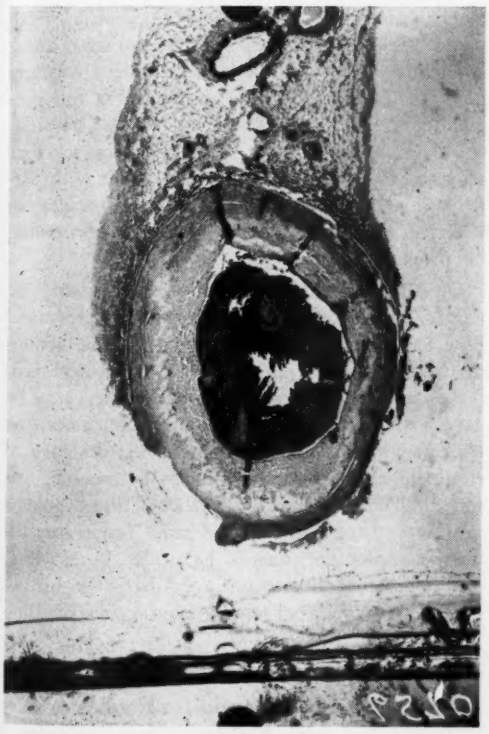

Figure 4. Photomicrograph showing embolus in the right coronary artery.

So this is a case of a patient who had multiple episodes of embolism over a period of many years, who terminally presented a coronary embolism with an infarct in the right ventricle and an embolism in the superior mesenteric artery with infarction of the entire small intestine and the right half of the large intestine.

Summary

A 48-year-old female patient long under observation for rheumatic heart disease and its common complications of congestive failure and auricular fibrillation and embolic episodes is suddenly seized with severe chest pain. This situation is immediately followed by coma and profound shock. The electrocardiogram accurately indicated a posterior myocardial infarction. Myocardial infarction due to arteriosclerotic heart disease, dissecting aneurysm, and coronary occlusion due to embolization were features of the differential diagnosis. The latter was the ante mortem clinical diagnosis considered most likely based upon the past history and the persistent comatose state.

To Study Effects of Radiation

A national survey to determine the prevalence of congenital malformations among the children of physicians, laboratory aides and others who are constantly exposed to radiation through their work in x-ray or radium therapy will be made soon with the aid of a Public Health Service grant.

The survey, to be made among some 4,000 radiologists in the United States, will be the first attempt to determine in a human population whether and how radiation causes hereditary changes within a period of one or two generations. It may also provide information on radiation effects which may result from atomic warfare, revealing the extent of safety provided by current protective measures.

The University of Colorado School of Medicine, Denver, announces a series of postgraduate courses open to all physicians who are members of their respective county medical societies. The following courses are scheduled: Chest Diseases, October 18-20; Heart Disease, November 15-17; Poliomyelitis, December 13-15; Neuro-Anatomy Seminars, Thursday evenings, August 16-December 27; Pathological-Physiological Seminars, Tuesday evenings, September 24-December 15.

Inquiries may be directed to the Office of Director of Graduate and Postgraduate Medical Education, 4200 East Ninth Avenue, Denver 7, Colorado.

BLUE SHIELD

In a recent article in *Medical Economics* written by a doctor using a pseudonym, on the subject of hospital-physician relations, Blue Cross and Blue Shield were brought into the picture to a considerable extent. Some of the main implications were as follows:

1. Hospitals are using Blue Cross as one of their tools to dominate the practice of medicine. According to the anonymous author this is done by writing into Blue Cross contracts benefits for medical services such as x-ray, anesthesia and pathology.

2. Doctors have little, if any, voice in Blue Cross policies.

3. Blue Shield is under the domination of Blue Cross for the same general purpose. He implies that joint administration of Blue Cross-Blue Shield inevitably means Blue Cross contracts.

4. Health Service, Inc., a Blue Cross-owned national corporation, was also established to fit in with the long range objectives of hospitals. No mention was made of the fact that Blue Shield is also developing its own national corporation as an autonomous organization to offer prepaid medical care to national employers.

It is important to get the record straight as far as Kansas is concerned. An organization should be judged by its actual practice and methods, and not by generalizations and inuendo imputing hidden motives. If some of the conditions described in the article exist in other places, they do not exist in Kansas.

Blue Cross was organized in Kansas in 1942 primarily through the initiative of the hospitals. However, the enabling legislation called for equal representation on the governing board of doctors, hospitals and public. Today one-third of the Blue Cross board are physicians.

The original Blue Cross contract was written after consultation with the Kansas Medical Society. It excluded any benefits for x-ray, the administration of anesthesia and pathology. The present Blue Cross contract contains no x-ray or anesthesia benefit. It does include pathological examinations, which were put into the contract after consultation and acceptance by the pathologists. Even now it is anticipated that as Blue Shield membership grows to be nearly the equal of Blue Cross, pathology will eventually be transferred into the Blue Shield agreement.

No action is taken by Blue Cross or Blue Shield without consultation and approval of the physicians in question. For example, when Kansas Blue Cross was asked to participate in national accounts such as U. S. Steel, the contracts for which contained x-ray and anesthesia benefits, the plans first went to the Kansas Radiological Society and the Kansas Society of Anesthesiology for approval. It was finally agreed that in such participation the benefits would be underwritten by Blue Shield in the form of a rider in the Blue Cross contract.

Blue Cross and Blue Shield in Kansas are jointly operated by a single administrative staff. This joint operation is carried out in accordance with a carefully written contract between the Blue Cross and Blue Shield boards. Both corporations maintain complete autonomy in the setting of policies and program. For example, the Blue Shield board has complete authority to decide all matters of fees, contract provisions, types of services covered, types of services not covered; in fact, any question which would affect the practice of medicine. The Blue Shield board is composed of 18 physicians and three laymen.

The benefits gained from joint operation are numerous. However, the main advantage lies in economic operation. With the cost of prepayment plans rising on almost all other counts, Blue Cross and Blue Shield in Kansas have been able to consistently lower the administrative expenses which are now under 10 per cent of total income.

In addition to the structures of organization which permit careful management and separation of medical matters from hospital matters, Blue Cross and Blue Shield in Kansas are carrying out a full program of communications with doctors and hospitals on all important questions. Through the relations program, full discussion is brought about with participating physicians and the hospitals to give an effective voice in stating their wishes concerning the programs.

On the basis that an organization should be judged by what it does, Kansas Blue Cross and Blue Shield submit their records and their actions to the fair-minded judgment of the medical profession and the hospital field. While others may be having their troubles, in Kansas we submit that the structures of our organizations are best designed to eliminate most of these problems before they get started. This is not to say that all of the problems in Kansas have been worked out. There are still many complex decisions to be made. But at least it is hoped that there is not a real basis for distrust in the motivation of either Blue Cross or Blue Shield.

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SEARLE RESEARCH IN THE SERVICE OF MEDICINE

ACTIVITIES OF MEMBERS

Dr. F. R. Croson, immediate past president of the Kansas Medical Society, recently suffered a heart attack and as a consequence has retired from active practice in Clay Center. Plans for the future are indefinite, but Dr. and Mrs. Croson expect to spend the winter in South America. Dr. Croson has announced the sale of his practice and equipment to his partner, Dr. Carl H. Ruff.

* * *

Dr. H. L. Kirkpatrick, Topeka, was speaker on two programs at Eskridge last month, one a meeting of the Kiwanis Club and the other a meeting of the Parent-Teacher Association in that city.

* * *

Dr. Fred S. Dozier, Herington, has been elected president of the board of education in that city.

* * *

Dr. C. V. Black, Pratt, became a fellow of the International College of Surgeons at a meeting held in Chicago last month.

Dr. Ben H. Buck, Jr., recently completed a residency at Hines VA Hospital in Chicago and is now associated with Dr. Willard J. Kiser, Dr. Leo K. Crumpacker and Dr. Harold S. Bowman in Wichita.

* * *

Dr. S. A. Anderson, Clay Center, has been appointed health officer of Clay County to fill the unexpired term of Dr. F. R. Croson, who resigned.

* * *

Dr. L. O. Forney, Hutchinson, was guest of honor at a recent luncheon given by physicians and pharmacists of the city in celebration of his 80th birthday.

* * *

Dr. H. L. Galloway, Anthony, began his 41st year in the practice of medicine in that city on September 1.

* * *

Dr. Ernest W. Crow, Wichita, is doing postgraduate work in internal medicine at the University of Pennsylvania. He will complete the work in June, 1952.

DEATH NOTICES

HARRIS W. MANNING, M.D.

Dr. H. W. Manning, 83, who retired from practice in Emporia a year ago, died there September 8 after an illness of several months. He was graduated from Rush Medical College in 1897 and served his internship in St. Luke's Hospital, Chicago. In 1901 he began practice in Eureka and remained there until 1919, when he moved to Emporia. He had taken postgraduate work in dermatology at Tulane University, and in Emporia he specialized in that field. Dr. Manning was a fellow of the American Academy of Dermatology and Syphilology and was an honorary member of the Lyon County Medical Society.

* * *

CHARLES ALFRED THOMAS, M.D.

Dr. C. A. Thomas, 74, an honorary member of the Montgomery County Society, died in Wichita September 11 after several months illness. After his graduation from Keokuk Medical College of Physicians and Surgeons in 1904, he practiced in Danville and Edna until 1918, when he entered the Army. When he returned from service in France, he took postgraduate study in eye, ear, nose and throat work and practiced that specialty in Coffeyville for 20 years. In 1939 he moved to Wichita to join the medical staff of the Boeing Company, and later was on the staff of the VA Hospital in that city. He retired from that service two years ago.

* * *

WILLIAM TILTON RICH, M.D.

Dr. W. T. Rich, 45, who had closed his office in Neodesha a year ago because of heart disease, died at the Wilson County Hospital on September 14. He was a graduate of St. Louis University School of Medicine with the class of 1932, and received his Kansas license in 1933. He served in the Navy during World War II and recently was commander of the third district of the Kansas Department of the American Legion. He was an active member of the Wilson County Medical Society and of the International College of Surgeons.

* * *

ALEXANDER HODGE BRESSLER, M.D.

Dr. A. H. Bressler, 79, an honorary member of the Pottawatomie County Society, died September 18 after having suffered a stroke the day before. He was graduated from the University of Louisville School of Medicine in 1894 and received his Kansas license in 1901. He practiced in Nickerson, Raymond and Manhattan, and before World War II retired to a farm near Wamego. He opened an office in Wamego during the war and continued to practice there until his death.

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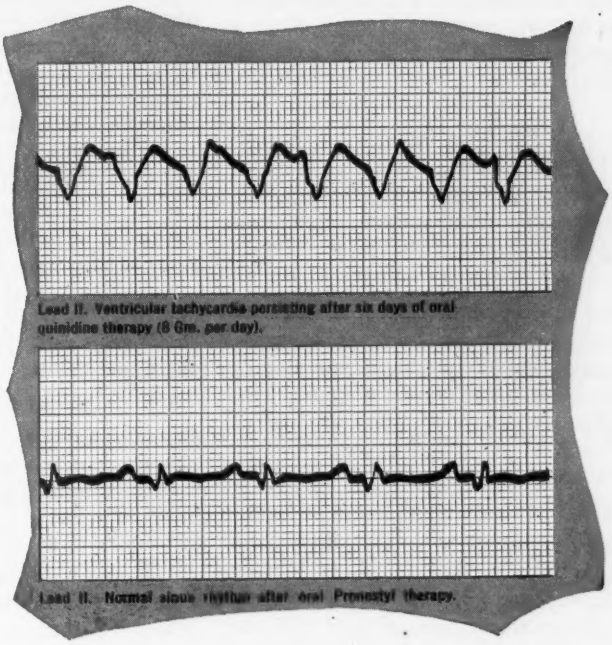

Lead II. Ventricular tachycardia persisting after six days of oral quinidine therapy (6 Gm. per day).

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Oral administration of Pronestyl is indicated in ventricular tachycardia and runs of ventricular extrasystoles. Intravenous administration is sometimes used in ventricular tachycardia and to correct ventricular arrhythmias during anesthesia. For detailed information on dosage and administration, write for literature or ask your Squibb Professional Service Representative.

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SQUIBB MANUFACTURING CHEMISTS TO THE MEDICAL PROFESSION SINCE 1858.

Dr. James Mott, who resigned as director of the health department for Lawrence and Douglas County in July, has been named director of the Division of Epidemiology of the Kansas State Board of Health, Topeka.

* * *

Dr. John G. Hoffer, Jr., formerly of Wichita, has completed a residency at the Fargo, North Dakota, VA Hospital and is planning to return to Kansas to establish a practice in Medicine Lodge.

* * *

Dr. J. Stanley Reifsneider, Wichita, showed pictures of his boat trip down the Colorado River to the Pratt County Medical Society at a meeting held September 12.

* * *

Dr. Preston J. Shafer has completed a residency at Wesley Hospital, Wichita, and is now associated in practice with Dr. J. W. Shaw in that city.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the Journal reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.

Excessive Fees

A prominent surgeon declares for publication that some surgeons actually charge too much, perform needless operations and split fees. Only a very small segment of American surgeons are guilty of these practices, Dr. I. S. Radvin said at the American Medical Association meeting at Atlantic City, but by doing so they threaten "our great heritage."

By that he meant the heritage of an excellent reputation built up over the years by American doctors. But like the bad apple that spoils the entire basket, the medical profession may suffer from the acts of a few individuals. Socialized medicine advocates are quick to exploit them.

Doctor Radvin himself gives some stern advice to his brothers in his speech in which he decries excessive fees, "ghost surgery," the hiring of unneeded consultants and the over eagerness of some surgeons to operate when operations are not necessary.

But such professional excesses are not apt to be alarming so long as members themselves are willing to face the truth and to discipline one another as Doctor Radvin has done.—*Kansas City Kansan, June 18, 1951.*

Fadeout May Be Temporary

Compulsory health insurance, sometimes known as socialized medicine, may still officially be part of the administration's program, but less is heard of it.

Possibly the fact that 11,000 organizations have officially gone on record against the proposal has something to do with the silence. These organizations are said to include leading national groups representing agriculture, labor, industry, veterans, university women and others.

The American Press, a magazine which is concerned with the problems and activities of weekly newspapers, says that "greater support was shown for the American Medical Association's campaign to fight government medicine than ever has been shown in any other national campaign—including government wartime campaigns."

Another angle in the socialized medicine situation is the fact that 72,000,000 persons now are enrolled in voluntary health insurance plans. They are taking care of their own requirements without government prodding.

Looks like federal medicine is dormant for the time being, but the planners probably will make it up at some future date.—*Dodge City Globe, August 30, 1951.*

* * *

Those Medical Ethics

A physician is being hauled upon the ethical carpet by his medical society for saying that the recovery of a woman's eyesight was as near a miracle as anything he has ever seen.

And once again the nation is treated to a look at what a part of the medical profession brands as unethical.

The county group thinks maybe the physician reaped a little "unethical advertising" from the quotation. In other words he got his name in too many papers.

It seems to *The Telegram* that if the doctor thought the recovery of the woman's sight was due more to God's ability than his own, he was being honest in not claiming credit and should be complimented for that honesty.

Or do the doctors just consider miracles as unethical generally?

The medical profession admittedly has a hard time keeping its own house swept clean. The business of healing and operating is so complicated that it offers a fine field of harvest for the quack.

But the business of policing a profession can get down to some very sorry hair-splitting sometimes. This appears to be one of those times.—*Garden City Telegram, September 12, 1951.*

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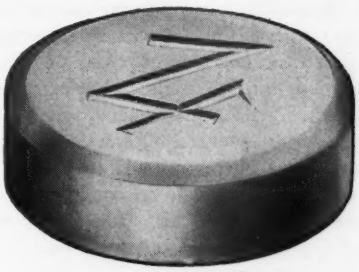

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Senior Thesis from the University of Kansas Medical School*

Potassium

Carl C. Gunter, M.D.**

Kansas City, Missouri

In the past few years we have gained a clearer understanding of the significance of potassium in health and disease. The purpose of this paper is to summarize the recent work with respect to this ion with primary emphasis on the (1) physiology of potassium, (2) clinical manifestations of potassium deficit, (3) clinical manifestations of potassium excess, and (4) discussion of a few of the more common clinical conditions affecting the level of body potassium.

Physiology of Potassium

Though it has been known that the cell membranes are freely permeable to water, dextrose, oxygen, carbon dioxide, urea and other nutrient and excretory products, it was believed in the past that they were impermeable to sodium, potassium and other electrolytes. That is how the great difference in concentration of these ions on the two sides of the cell membrane was explained. Ideas on this subject have been sharply modified in the last few years, and this modification has led to a much better understanding of the clinical physiology of potassium and to better therapy of dehydration.

The average concentration of serum potassium in normal human subjects is about 4.7 milliequivalents per liter with a normal range from 4.0 to 5.4 milliequivalents per liter. In the intracellular fluids, the concentration of potassium is much higher, for here potassium is the principal cation, the cellular sodium concentration being low. Recent animal experiments[1] show that the intracellular concentration of potassium is 23 times greater than that of the serum. The total potassium of the adult body is about 160 grams or 4000 milliequivalents. Of this total amount only about 70 milliequivalents is in the extracellular fluids.

Studies with radioactive isotopes of sodium and potassium and with heavy water have shown that the cells are permeable to sodium and potassium. These elements permeate cell membranes slower than water does. When heavy water was injected intravenously, equilibrium with the total body water was established 120 minutes later. When radioactive sodium was injected, equilibrium with extracellular sodium was reached in 60 minutes but not with the total body sodium until 24 hours. Radioactive potassium requires 15 hours before reaching equilibrium.

There is no adequate explanation of how and why the tissue cells acquire and maintain so much higher a concentration of potassium than that of the serum or interstitial fluids. Since it has been repeatedly demonstrated that potassium leaves the cells during potassium losses from the body[2] and enters cells during its administration,[3] it appears that there is normally an equilibrium being maintained between the low extracellular concentration and the high cellular concentration.

This equilibrium is unstable, and it is reasonable to believe that this unstable equilibrium is being maintained by the oxidative energy of the cells. If this be so, then cellular anoxia would tend to send potassium out of the cells into the extracellular fluids. Such a phenomenon might account for the rise in serum potassium concentration with exercise and might explain the progressive transfer of red cell potassium to plasma in stored blood.

It has been estimated[4] that, on a normal diet, the potassium intake is about four grams per day. The daily intake of certain foods rich in potassium is quite essential for the maintenance of a normal serum potassium. Potassium balance is preferably maintained by dietary oral intake; however, in some cases it is necessary to give the potassium parenterally.

The commonly used foods that are especially rich in potassium are potatoes, which contain more than 400 mg. per 100 grams. Other foods that contain more than 300 mg. per cent of potassium are meats, fowl, fish of all kinds, sweet potatoes, peas, beans, beets, cabbage, celery, chard, chestnuts, spinach and other greens, pumpkin, squash, parsnips, dried fruits, figs, prunes, banana, pineapple, nuts of all kinds, bran and molasses.

Soon after a meal the serum potassium rises slightly but then falls to its previous level. In the normal adult the excess potassium is excreted in the urine. In gastrointestinal disease not only is the potassium of the secretions lost but also that which is ingested. The kidneys continue to excrete potassium, and thus a deficiency state develops.

The equilibrium between the intracellular and the extracellular fluid is constantly being interfered with by events which unduly raise or lower the

*This is one of 11 senior theses selected for publication by the Editorial Board from a group of 15 judged the best by the faculty of the University of Kansas School of Medicine.

**Thesis written while the author was a senior student at the University of Kansas School of Medicine. Dr. Gunter is now serving his internship at General Hospital, Kansas City, Missouri.

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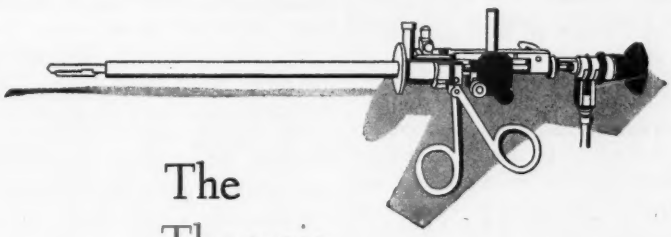

The Thoracic Surgeon . . .

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serum potassium. The functions take place at a faster rate than the slow permeability of the cell membrane would allow. Important among these changes is the continual excretion of potassium from the body by a renal mechanism designed for removing the excess potassium rather than conserving it during deficiency. The renal losses are exaggerated by diuresis, acidosis, and excess adrenocortical hormone.

In starvation, trauma and disease, cellular breakdown occurs with the potassium of these destroyed cells being added to the extracellular fluid. Sometimes it is added at a rate greater than can be excreted or transferred to cells, and an excess of potassium is then found in the serum. There is a definite ratio between the nitrogen breakdown and the amount of potassium released. For every gram of nitrogen broken down, 2.4 grams of potassium are released.

Expansion of the extracellular fluid compartment in the treatment of dehydration by intravenous administration of potassium-free fluids produces a rapid lowering of the serum potassium concentration, because of dilution of the extracellular compartment by the injected fluid, influx of water from cells to lower the osmotic pressure of the interstitial fluid and raise that of the cellular fluid as well as increased excretion of potassium.[5] When the cells have already lost much water, some of the transfer of water from the cells is ultimately annulled by transfer of sodium into the cells.

The concentration of potassium in the extracellular compartment is clearly the resultant of how much of this element comes in and how much goes out. Potassium can enter this compartment by way of the intestinal tract either by absorption of ingested potassium or by emerging from the cells. Potassium can leave the extracellular space by entering the cells or by excretion in the urine. Only in diarrheal states are significant amounts of potassium lost by way of the stool. Potassium is present in the gastrointestinal fluids in quantities as great as 40 milliequivalents per liter in the gastric juice and 8-10 milliequivalents per liter in the intestinal juice. Normally almost all of this potassium is reabsorbed through the mucosa and into the bloodstream to the extent that only about 10 milliequivalents are lost in the feces per day.

In diarrhea and vomiting 10 to 20 times this amount is lost daily. Howard and Carey[6] also found that gastric juice and juices of the small intestine as well contain potassium in concentration severalfold greater than the plasma. This was true in patients with and without free hydrochloric acid, and it has been only in patients suffering from very low potassium levels that gastric juice has contained less than 10 milliequivalents per liter. Usual amounts are from 15 to 25 milliequivalents per liter. Thus excessive vomiting or prolonged gastric suction might prove to be ways of losing considerable quantities of potassium.

Homeostatic mechanisms are so delicately arranged that it requires a major functional disturbance to alter the concentration of potassium in the extracellular fluids from its normal range. When elevation or depression of the potassium concentration does occur, however, serious consequences ensue.

This continued excretion of the ion in urine is an important if not the principal factor in producing the cellular deficit. This continued renal excretion of potassium does not indicate abnormal renal function, but rather normal function of kidneys under abnormal circumstances. Concentration of urine potassium below four milliequivalents per liter has been recorded during diuresis and potassium depletion. Concomitant serum concentrations in one study were not greater than those in urine. In several series the concentration of potassium was never lower in urine than in serum.

Considering renal tubular function as a whole and assuming that the tubular cells are surrounded by the usual type of extracellular fluid, no evidence has been found that potassium can be reabsorbed against a concentration gradient. This does not exclude the possibility that such reabsorption may occur in a given portion of the renal tubule.

Clinical Manifestations of Hypopotassemia

Since in almost all instances of hypopotassemia there is pronounced cellular deficiency of potassium, many of the symptoms may be due to the latter rather than to the low serum levels.[7] This is not absolutely true because in the paralysis of familial periodic paralysis there is a shift of the extracellular potassium into the cells and the symptoms are due to the low serum potassium level.

In most cases, however, the clinical manifestations associated with low serum potassium levels are probably produced by the cellular deficiency in potassium. Unfortunately, these symptoms are not specific and are frequently found in many debilitating conditions. The most prominent symptoms are extreme muscular weakness, anorexia, lethargy, distention of the intestine, myocardial degenerative changes and edema of the lungs.

The electrocardiographic observations associated with low serum potassium concentrations are progressive lowering and broadening of the T wave and lengthening of the Q-T interval. The S-T segment usually becomes depressed. As the serum potassium falls still further a U wave may make its

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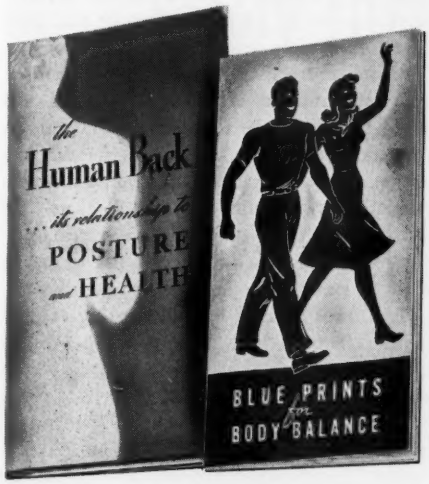

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appearance. These findings are probably directly due to the low extracellular potassium concentration.

Abnormally low levels of extracellular potassium can, on theoretical grounds, result from: (1) dilution by low potassium fluids, (2) losses of the cation in urine or other body fluids, or (3) transfers of potassium into the cells.

Clinical Manifestations of Hyperpotassemia

Symptoms of hyperpotassemia chiefly involve the heart. Paresthesias of the extremities and flaccid paralysis have been reported,[8] but in most instances the cardiac observations are the only significant ones. There are characteristic electrocardiographic changes.[9] The T-wave becomes increasingly taller with a narrow base; this can be seen with serum potassium levels of 7.5 milliequivalents per liter. With higher levels, as the T-wave becomes even higher, the QRS complex becomes progressively broader, indicating intraventricular conduction defect. The P-wave may disappear. Ultimately the heart stops in diastole. Death can occur when the serum level reaches 10 milliequivalents per liter, but reversible changes have been known to occur even at higher levels.

The electrocardiographic changes are seen to occur in a fairly regular sequence and at fairly definite ranges of serum potassium. First to appear is an increase in the amplitude of the T-wave, usually accompanied by a shortening of its duration. This change becomes apparent at concentrations of 5.0 to 7.8 milliequivalents per liter, and increases progressively until the T-wave equals the QRS complex in amplitude and brevity. Somewhat later, at concentrations of 7.8 to 9.0 milliequivalents per liter, the S-T segment begins to sink below the isoelectric line and continues to drop as the concentration rises. As the concentration of serum potassium reaches 10.0 to 12.0 milliequivalents per liter, the QRS complex becomes definitely wider. When the concentration reaches 14.0 to 16.0 milliequivalents, which is a definitely lethal concentration, there is almost complete disorganization of the complex. The P-waves first begin to widen and lose amplitude at concentrations of 9.4 to 11.0 milliequivalents per liter and then quickly disappear as the concentration rises higher. No changes are noted in the P-R interval up to the time of extinction of the P-wave. Intraventricular block begins at concentrations of 10 milliequivalents per liter and cardiac arrest takes place at from 14.0 to 16.0 milliequivalents per liter. Usually no ectopic arrhythmias of any type occur unless the rate of serum potassium rise is extremely rapid.

It is important to recognize that these electrocardiographic changes are due entirely to the extracellular accumulation of potassium. The intoxication may take place even in the presence of severe cellular deficiency of potassium. That is why it is important to administer potassium slowly and preferably via the stomach, even in pronounced deficiency; extreme care is required when renal function is too poor to permit rapid adjustment of serum potassium concentration.

Potassium in Diabetic Acidosis

The three factors contributing to the abnormally low levels of extracellular potassium that were previously mentioned are probably operative in diabetic acidosis and coma. Such patients usually develop anorexia and vomiting during the early phases of illness. In addition there is an accompanying polyuria. As a consequence the intake of potassium is reduced essentially to zero. At the same time considerable amounts of potassium are lost in the urine. Even though the body stores of this electrolyte are considerably depleted in these various ways, the patients are frequently admitted with elevated or normal concentration of potassium in the serum. This is explained by the attendant contraction of the plasma and extracellular volumes as a result of dehydration. The dehydration is not only extracellular, accompanied by sodium and chloride loss, but is intracellular with potassium and phosphate loss.

The hypopotassemia in the past has been aggravated by our methods of treatment of diabetic acidosis. Treatment in the past has been to give insulin, isotonic sodium chloride solution and alkali either as sodium bicarbonate or as sodium lactate. In most instances in which the patient is dehydrated and acidotic to the point of collapse, this treatment is satisfactory in that it restores the patient to consciousness. However, in the less severe cases there is a fall in the potassium level as the extracellular volume is expanded. Along with the increased extracellular volume, there is an increase in renal function which produces a further loss of potassium. At the same time potassium moves into the cells under the impetus of insulin, restoration of carbohydrate catabolism, glycogen deposition and protein formation. Gluconeogenesis carries potassium from extracellular fluid into the cells. This process occurs in specialized tissue that contains glycogen, such as liver and muscle. It is increased by glucose ingestion, by insulin and epinephrine.

The fall in serum potassium levels is rapid but temporary. In the normal person the change in serum potassium concentration is negligible, but in such potassium deficient persons as those with diabetic acidosis, the rapid decline of an already low serum potassium concentration may produce serious symptoms. The consequence of all of these factors is an abrupt fall of serum potassium to a level pro-

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ducing respiratory paralysis and cardiac damage.

This catastrophe can be avoided in many instances by the judicious use of potassium chloride along with sodium chloride and sodium lactate. The potassium chloride should be given preferably by mouth or stomach tube, two grams at a time. It may be given successfully by proctoclysis. If given intravenously, a liter of fluid should not contain more than two grams of potassium chloride.

In treatment of patients with diabetic acidosis, it is of utmost importance to keep in mind the possibility that symptoms of extreme muscular weakness and rapid shallow respiration are due to a low serum potassium. This is especially true when treatment is characterized by the administration of large amounts of glucose, insulin and fluid, because these are circumstances which favor the rapid deposition of liver glycogen and the establishment of diuresis. Both glycogenesis and diuresis have been shown to remove potassium from the serum.[10]

The striking retention of potassium administered to a series of patients points to the existence of concomitant deficits of this cation in the body cells.[11] This retention was not noted when potassium was given by mouth to healthy adults. Presumably such subjects do not have potassium deficits and hence the administered potassium is promptly excreted. Diabetic patients in acidosis or coma are, however, depleted of potassium.

The hypopotassemia of extracellular fluid which develops during treatment on a potassium-free regimen stems from a number of sources. It is obvious that it results in part from expansion of body water with the administration of fluids, and in part from a continued loss of potassium in urine.

The physiological significance of these extensive potassium deficits can be as yet partially and only indirectly evaluated. It is known, of course, that muscular paralysis and abnormally low levels of extracellular potassium frequently coexist. This is not, however, an invariable association since paralysis can occur with normal serum concentrations of potassium, and data of Danowski et al[11] indicates that pronounced hypopotassemia can occur without evident paralysis.

At present, only analogies can be drawn as to possible deleterious effects of the deficits of intracellular potassium. It has been shown, for example, that the depletion of cellular potassium produced by over-dosage with desoxycorticosterone acetate or by restriction of potassium intake is associated with degenerative changes in the myocardium.

Holler[10] feels that respiratory paralysis seen in the treatment of diabetic acidosis on a potassium-free regime is a rare occurrence and that unusual conditions must necessarily be present before such a clinical picture is seen.

Postoperative Hypopotassemia

A syndrome occurring in patients after operation has been described by Eliel et al.[12] It is characterized by clinical manifestations of apathy, lethargy, nervousness and irritability, muscular weakness, abdominal distention and occasional ileus, occasional confusion, disorientation, delirium, muscular twitching and tetany, electrocardiographic changes and occasional arrhythmias and edema; blood chemical findings of hypopotassemia, metabolic alkalosis, hypochloremia, hypoproteinemia and, often hypophosphatemia; and by prompt reversal of the clinical and blood chemical abnormalities upon administration of adequate amounts of potassium.

The syndrome developed primarily in patients who were being fed parenterally and who received little or no potassium. Intracellular potassium deficit was demonstrated by balance studies on patients in whom the syndrome developed after operation. The deficit arises as a result of renal loss. Losses of potassium from intestinal fluids have been small or absent in most cases reported in spite of the fact that gastric and intestinal juices contain a significant amount of potassium as mentioned previously.

Known factors in the pathogenesis of postoperative potassium loss are potassium deprivation, loss of gastrointestinal secretions, large infusions of glucose or saline solution, dehydration and malnutrition. These factors do not adequately account for some of the potassium losses observed.

Evidence is available that adrenal hyperfunction resulting from surgical trauma leads to potassium deficit; the clinical and electrolyte disturbances seen in the syndrome resemble those described in some cases of Cushing's syndrome; metabolic alkalosis and potassium deficit have been produced frequently in human beings by the injection of adrenocorticotrophic hormone and cortisone; and there is a striking resemblance between other metabolic changes seen postoperatively and those associated with adrenal hyperfunction. It is possible that adrenal hyperfunction resulting in potassium deficit may occur after any type of tissue injury.

Therapy in these cases is directed toward replacing the potassium deficit. Death after *injudicious* administration of potassium is not uncommon. Cardiac arrest from heart block may occur in human beings at serum levels of about 10 milliequivalents per liter. Potassium therapy should therefore be undertaken only with an understanding of the requirements, safe methods of administration and contraindications.

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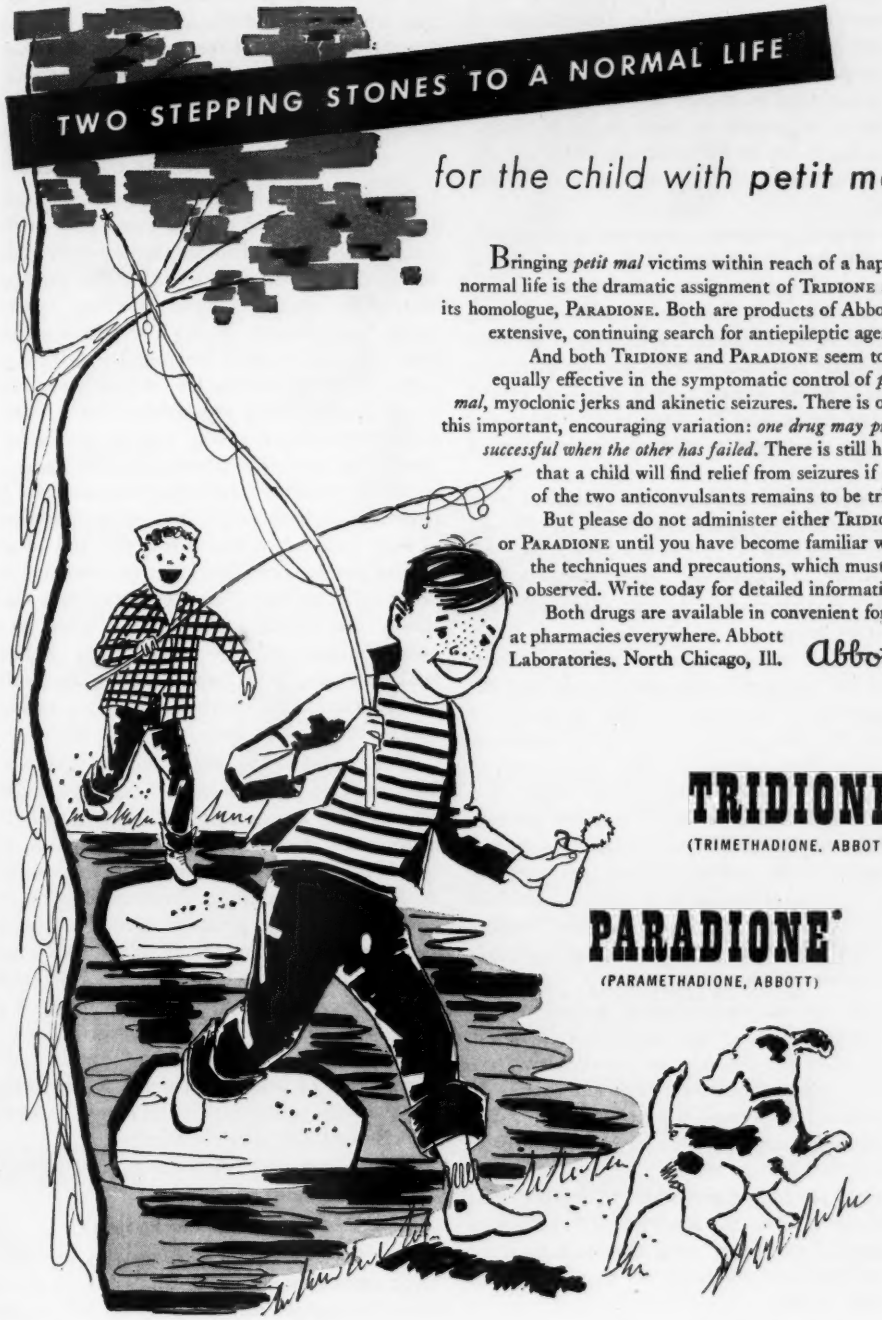

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alents of potassium after relatively atraumatic procedures prevents potassium depletion in patients being maintained on intravenous administration of fluids. After more traumatic procedures, the daily requirement can usually be met by the provision of 80 to 100 milliequivalents. This amount is ordinarily adequate for repair of deficits also, since urinary losses tend to decline as the deficit grows. Replacement of potassium losses in most gastrointestinal fluids can be estimated adequately on the basis of 10 milliequivalents potassium lost per liter of fluid.

Since urinary potassium losses are greatest during the first few postoperative days, early provision of potassium is indicated in patients who cannot eat and who must be fed parenterally. It should, however, be withheld during the first 24 to 48 hours postoperatively and from patients who are extremely dehydrated, whose urinary output does not exceed 500 cc. per day or who demonstrate nitrogen retention, unless the serum potassium concentration is low.

The forms of oral and parenteral potassium most commonly used are: capsules or potassium chloride, one gram every four hours (80 milliequivalents per day), which are taken with ample water or fruit juice; and potassium chloride ampoules, each containing 1.49 grams (20 milliequivalents) in 10 cc. of water, which can be added to glucose or saline solutions for parenteral administration in any desired quantity at the bedside. Two ampoules of potassium chloride per liter, providing a concentration of 40 milliequivalents per liter, are usually recommended. This can be administered safely at a rate of 80 to 100 drops per minute. In the presence of serious complications from potassium deficit, a concentration of 80 milliequivalents per liter can be used. Electrocardiograms as a guide to judicious potassium repletion must be emphasized, particularly if determinations of serum potassium levels are not readily available.

Younger patients excrete more potassium than older ones undergoing the same operation, and those who have had the more serious operative procedures, e.g., abdominoperineal resection, excrete 73 per cent more free potassium than the controls who did not undergo any operation, or those subject to herniorrhaphy.

The need for adding potassium to postoperative fluid therapy rests on the assumption that losses of this element should be replaced. On this basis about one or two grams of potassium salt should be added to the daily intake.

The significance of the potassium losses may be discussed in terms of protein losses. This is usually done by establishing a K:N ratio based upon computing the potassium loss in millimoles and the nitrogen loss in grams. A ratio of three or less is considered by most authorities to represent the normal relationship of intracellular potassium to protein. The findings of Elman[5] and associates suggest that the destruction of the patient's protoplasm was not the sole source of the potassium loss, and that much of it originated in another way, perhaps by loss of cell potassium without loss of protoplasm.

Hypopotassemia in Intestinal Obstruction

Balance studies similar to those conducted in diabetic acidosis and diabetic coma have served to define, in part, the chain of events which results in low serum potassium values in infants with pyloric obstruction and prolonged vomiting. Although actual measurements during the prehospitalization phase of such subjects are not available, it seems highly probable that the loss of potassium in vomitus is of considerable magnitude. This is, of course, accompanied by a greatly reduced intake of potassium in food. On admission these patients are frequently found to have hypopotassemia and hypochloremia, high serum bicarbonate levels and deficits of body water. It is true that at the time of admission or shortly thereafter, losses of potassium in the urine are quite low. This does not exclude the possibility that earlier losses via this route were of greater magnitude. The usual therapy of low potassium fluids, while oral feedings are withheld, either produces or further aggravates hypopotassemia.

Then potassium is lost from the body via the kidneys or the gastrointestinal tract. A good portion of it is accompanied by chloride, which has come from the extracellular fluids. This unbalanced loss of extracellular anions is compensated for by a rise in extracellular bicarbonate. This is apparently why acute potassium deficiency is usually associated with alkalosis.[13] If such alkalosis is treated by administration of sodium chloride solutions, more potassium is lost, if renal function is good, along with the chlorides; the alkalosis tends to persist, and edema is produced. On the other hand, administration of potassium chloride raises intracellular potassium concentration and that of extracellular chloride and dissipates the alkalosis.

Similar losses are reported in persistent infectious diarrhea of infants.[14]

Familial Periodic Paralysis

It has been known for many years that the paralytic manifestations of this disease were related to potassium metabolism. As early as 1905, potassium bromide was being used effectively in the treatment of the paralytic state, but the effect was attributed to its sedative action. Many observers have recorded

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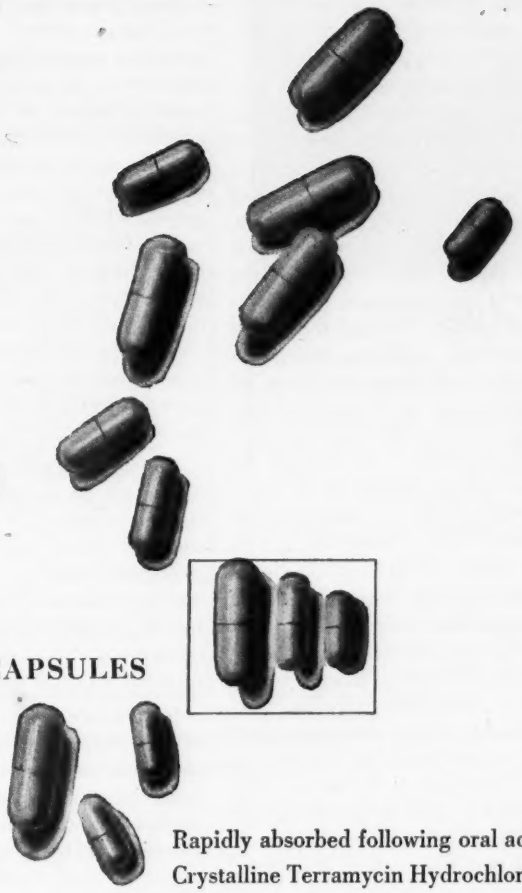

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the level of potassium in the blood in this condition and have found it to fall as much as 30 per cent while in mild attacks the fall may be only 5-10 per cent.

This is a rare disease affecting males more frequently and characterized by the cardinal symptoms of periodic paralysis, loss of reflexes, and loss of electrical excitability of the muscles. The onset is sudden, usually at night, the paralysis beginning peripherally in the legs and traveling centrally. It is usually symmetrical but it may be unilateral. The paralysis is usually complete, flaccid, and involves the body musculature. Consciousness is not affected.[15] The electrocardiographic changes during an attack of familial periodic paralysis have been studied, and the most striking changes and those responsible for the bizarre form of the electrocardiogram were prolongation of the PR interval, QRS, and the Q-T intervals, alterations in the form of the R-T segments, and decrease in amplitude of the T waves. These electrocardiographic changes are associated with reduction in serum potassium.

Attacks of paralysis have been induced in patients with periodic paralysis by the ingestion of 300 grams of glucose in hypertonic solution. The development of these attacks was accompanied by falling serum potassium levels and was unassociated with concomitant excessive potassium excretion. Recovery was associated with a rising serum potassium level. In some cases, a comparable but less marked and less sustained fall and rise in the serum phosphorus level is observed. Also noted during the phase of maximal paralysis was a suppression of both phosphorus and potassium urinary excretion.

Evidence is also available which supports the hypothesis that hepatic intermediate carbohydrate metabolism plays an important role in periodic paralysis by lowering the sugar content of muscle. It is felt by Gass[16] and associates that the serum levels of potassium in periodic paralysis do not directly determine the onset of paralytic manifestations.

Potassium in Chronic Nephritis

In renal failure various authors have noted an accumulation of body potassium, and some have noted concomitant alterations in the electrocardiogram. The electrocardiographic changes accompanying a rise of serum potassium are an increase in the amplitude of the T waves. This is followed by an impairment of intraventricular conduction and cardiac arrest when the serum potassium reaches about 10 milliequivalents per liter.

A search of the literature has not revealed instances of chronic nephritis associated with symptoms and electrocardiographic changes resulting from excessive loss of body potassium. Brown[17] and associates were the first to report cases of hypopotassemia in chronic nephritis. Although the serum potassium was not determined in all of his cases, the response to potassium chloride with the resulting rapid subsidence of the paralysis seems sufficient evidence to justify the deduction that a disturbance of body potassium was the cause of the paralysis observed in his cases. The transient electrocardiographic changes associated with the attacks contribute further evidence of a disturbed body electrolyte balance. The changes seen, particularly the low T waves and impaired auriculoventricular conduction, are those which are also seen in patients who have a low serum potassium from other causes. The return of the electrocardiogram to normal after the administration of potassium chloride by mouth was striking.

It should be remembered, however, that in patients with pronounced renal insufficiency, particularly when there is a decrease in urinary output or complete anuria, the kidney has difficulty in disposing of potassium, and that potassium poisoning may be produced by the administration of potassium salts in abundant quantities. This may occur spontaneously and may reach a degree of intoxication sufficient to cause cardiac arrest.

Cardiac arrest due to an increase in the potassium of the serum to toxic levels has not been demonstrated in uremic patients who have maintained a large volume of urine up to the time of death. Potassium intoxication in patients with uremia is spontaneous and results from oliguria or anuria and failure of renal excretion. The three sources from which the high concentration of potassium in the serum appears to be derived are diet, red cells introduced by transfusion of blood, and tissue metabolism. Of these, tissue metabolism alone is sufficient to cause death in cases where there is marked oliguria or anuria. It has not been possible to demonstrate by chemical methods alone that potassium may be an actual cause of death from uremia in man.

Discussion

A discussion of the recent advances in the knowledge of the potassium ion is given along with a presentation of some of the disease conditions in which the serum level of the ion is affected. No attempt has been made to cover all of the disease states in which the metabolism of potassium is affected; only a few are discussed along with their treatment. Similar mechanisms that function in the described disease states also operate in other diseases. A brief discussion of methods of potassium administration is given.

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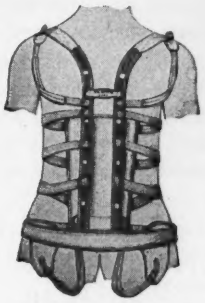

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BOOK REVIEWS

Let's Have Healthy Children. By Adelle Davis. Published by Harcourt, Brace and Company, New York. 314 pages. Price $3.00.

The author is a consulting nutritionist. She has studied at Purdue, the University of California, Columbia, and the University of California at Los Angeles. She received a Master of Science degree from the University of Southern California Medical School.

The book is intended to be a guide for mothers and expectant mothers. It covers the subject of motherhood and child care through the pre-school age. It is written in such a way that it should be easily understood by the layman.

Her references are many and the sources are excellent. Many of her statements of fact regarding certain foods, I am sure, would be revealing to many physicians. She very ably discusses the problems of breast feeding, formulas, thumb sucking, the introduction of solid foods and feeding problems. Every physician who cares for infants and children would do well to read this part of the book.

Any mother who reads this book will most certainly come to her physician with many questions. I think this is good. There are many controversial subjects but generally speaking it is a book of well founded facts and is well worthwhile.—*J.R.P.*

* * *

Let's Cook It Right. By Adelle Davis. Published by Harcourt, Brace and Company, New York. 626 pages. Price $3.00.

"Good Health Comes from Good Cooking," the sub-title of this book, illustrates the author's purpose, namely, promoting the consumption of foods higher in vitamins and minerals. The roasting of meats at low temperatures and the cooking of vegetables in very little water are two points emphasized. The use of black strap molasses, powdered milk, and whole wheat cereals is emphasized to the extent that one almost believes that all sugar should be replaced with black strap molasses, that all milk must be reinforced with powdered milk, and that the use of refined cereals is fatal.

Many statements sound exaggerated to the scientific person, but would be swallowed by the gullible because of the authoritative way in which they are written. No references are given to prove the assertion that "experiments have shown that most mouths are sterile after onions and garlic are chewed."

The book is written in an interesting, attention-getting manner. The first six chapters deal with menu planning, the use of seasonings, kitchen equipment, time saving ideas, and the psychology of introducing new foods. The manner of giving a basic recipe followed by variations should be an aid to new cooks. Principles underlying food preparation are given. Originality and variety characterize the recipes and menus given.

Eight chapters deal with meats, gravies, dressings, fish, and meat substitutes. Cuts of meat are clearly described. Other chapters tell how to prepare appetizers, salads, soups, vegetables, breads, cereals, milk drinks, fruits, desserts, and candy. Canning and pickling are discussed. One chapter tells where to get these "necessary" foods, such as brewer's yeast, yogurt, wheat germ, and soy flour, not commonly

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The Mechanisms of Cell Division. Edited by M. J. Kopac. Published by the New York Academy of Sciences, New York. 265 pages. Price $3.50.

This series of papers was originally presented at a conference of the New York Academy of Sciences. It gives a good cross-section of the numerous experimental approaches to the problems of cell division as presented by a distinguished group of investigators. The first papers discuss the classic methods, including studies of the cleavage and differentiation of marine eggs, cell division in tissue cultures, the behavior of mitochondria and Golgi substance in dividing cells, and the fission of amoeba. Physical methods such as microdissection, hydrostatic pressure, and centrifugation are considered next.

Then pharmacological studies are described. Among the drugs and chemical substances whose effects are reported are colchicine, substituted phenols, nitrogen mustards, podophyllin, and other mitotic inhibitors.

The next two papers are on the effects of irradiation on dividing plant and animal cells. The final paper is a theoretical discussion of the probable ultrastructures of protoplasm (molecular aggregates, small particles and fibrils) and their possible relationship to the mechanics of cell division.

This collection of papers is highly recommended. It shows clearly the wide variety of disciplines requisite to modern experimental cytology. Although many of the papers are short, the bibliographies are in most cases extensive. Anyone interested in pursuing one or more aspects of the research on cell division would do well to start with this publication. It is a coherent, stimulating and well done piece of work.—*M.E.W.*

State Hospital Out-Patient Department

The Topeka State Hospital, serving a 27-county area, announces a new source of referral for psychiatric patients in its recently organized out-patient department.

Designed to provide treatment for patients who cannot afford private psychiatric care, before they become so ill they need hospitalization, the department has a graduated scale of fees in accordance with the patient's income. For diagnostic and evaluation examinations the average fee is $1.00 an hour. For psychotherapy, the fees range from 25 cents to $10 a visit. Average fees range between $2.00 and $3.00 a visit. Patients who can afford more than $10 a visit are referred to private facilities.

Physicians may refer patients for consultations and examinations. Where consultations are requested, a report is sent to the referring physician and the patient is re-referred to his physician. Where examinations are requested for the purpose of determining whether the patient might be accepted for psychotherapy in the out-patient department, a report of the examination findings and recommendations is sent to the referring physician. When psychotherapy is indicated, the patient will be carried in out-patient treatment, but will be referred to his own physician for physical treatment when necessary. Progress reports will be sent the referring physician on request.

Physicians wishing to refer patients under this program should write the Out-Patient Department, Topeka State Hospital, giving pertinent data on name of patient, age, marital status, chief complaint, duration of the problem, and a summary of psychiatric and medical status.

ANNOUNCEMENTS

The 19th annual assembly of the Omaha Mid-West Clinical Society will be held at the Hotel Paxton, Omaha, October 29-November 2. Nationally known guest speakers will present addresses and conduct clinics and question and answer periods, and members of the society will give lectures, panel discussions and scientific exhibits. A guest panel on blood dyscrasias is scheduled for Friday morning, November 2. Members of the American Academy of General Practice may receive credit, hour for hour, toward the 50 hours of formal postgraduate study required of them every three years.

Complete information may be obtained from the executive office of the society, 1031 Medical Arts Building, Omaha, Nebraska.

Flood Damaged Drugs Replaced

The destruction of pharmaceutical products as a result of the July flood in this area caused no financial loss to hospitals and pharmacies. All products were replaced without charge by the various firms which had sold the drugs.

Several pharmaceutical houses issued press releases to the effect that they were replacing lost or damaged drugs. A recent announcement from the organization of drug product salesman states that all firms selling in this area have adopted the same policy.

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Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal System	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular System	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references [3,4,5,6,7] given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Oculo-Cardiac Reflex Abnormalities
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives. [8,9,10]

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ABSTRACTS FROM CURRENT LITERATURE

NPH Insulin

NPH Insulin: Its Comparison with Previous Insulin Regimens. By H. M. Baganz, S. C. Carfagno, B. Y. Cowan and E. S. Dillon, Am. Jnl. Med. Sc., 221:1, 1-6 (July) 1951.

NPH insulin was perfected by Krayenbühl and Rosenberg in Hagedorn's laboratory. It differs from protamine zinc insulin essentially in its protamine content. Protamine zinc insulin contains 1.25 mg. of protamine per 100 units insulin, whereas NPH insulin contains approximately 0.5 mg. of protamine per 100 units of insulin. NPH insulin usually acts promptly enough to control the postprandial rise in blood sugar; it reaches its peak action in approximately eight hours and its duration of action is approximately 24 hours. This curve of action is similar to that of a 2:1 mixture of regular and protamine zinc insulins.

In this investigation of comparative actions, 41 patients were studied, on standardized quantitative diets, in a hospital environment, and with constant daily degrees of exercise. Each patient was first standardized and studied on protamine zinc insulin, protamine plus regular (given separately), or regular insulin alone. Comparison was then made by use of NPH insulin, basing comparisons on blood sugar studies four times each day, fasting, two hours after breakfast, 3:00 P.M., and 9:00 P.M.

The morning postprandial effect of NPH insulin was superior in all cases which had previously received only protamine zinc insulin, and as good in most cases which had received protamine zinc plus regular insulin.

The fasting, 3:00 P.M., and 9:00 P.M. blood sugar levels were generally better on NPH insulin than on previous regimens.

The following rule is given for transfer to NPH insulin from other insulins: if the total dose is 40 units or less per day, give an equal amount of NPH insulin. If the dose is more than 40 units previously, give a dose of NPH insulin equal to the protamine zinc requirement plus 80 per cent of the regular insulin dose.—*E.J.R.*

Three Grants to K. U.

Three grants for research on heart disease have been awarded the University of Kansas School of Medicine by the United States Public Health Service, according to a recent press release. The grants total $22,350, distributed as follows: to Dr. Paul W. Schafer and Dr. Hampton W. Shirer, $14,040 for the analysis of pulse wave forms in heart and great vessel of dog and man; to Dr. Sloan J. Wilson, $5,160 for clinical and basic research on various blood coagulation factors; to Dr. Parke H. Woodard, $3,150 for studies of allergic shock.

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